

**In the United States Court of Federal Claims**  
**OFFICE OF SPECIAL MASTERS**  
**No. 15-789V**  
(to be published)

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LINDSEY MARTIN *and* RAYNARD \*  
MARTIN, *as representatives of the estate of* \*  
I.R.M., *deceased*, \*

Petitioners, \*

v. \*

SECRETARY OF HEALTH AND \*  
HUMAN SERVICES, \*

Respondent. \*

\*\*\*\*\*

Chief Special Master Corcoran

Filed: May 8, 2020

Sudden unexplained death in  
children; *Althen* prong one; Innate  
immune response; Cytokines;  
Brain malformation; Expert  
competence; Seizure.

*Richard Gage*, Richard Gage, P.C., Cheyenne, WY, for Petitioner.

*Julia M. Collison*, U.S. Dep't of Justice, Washington, DC, for Respondent.

**DECISION DENYING ENTITLEMENT<sup>1</sup>**

On July 27, 2015, Lindsay and Raynard Martin filed a Petition under the National Vaccine Injury Compensation Program (the “Vaccine Program”<sup>2</sup>), on behalf of their deceased son, I.R.M., alleging that the Flumist version of the influenza (“flu”) vaccine he received on September 24, 2014, caused his death two days later. Pet. at 1-2 (ECF No. 1). A hearing in this matter was held on May 23–24, 2019.

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<sup>1</sup> This Decision will be posted on the United States Court of Federal Claims’ website in accordance with the E-Government Act of 2002, 44 U.S.C. § 3501 (2012). **This means the Decision will be available to anyone with access to the internet.** As provided by 42 U.S.C. § 300aa-12(d)(4)(B), however, the parties may object to the published Decision’s inclusion of certain kinds of confidential information. Specifically, under Vaccine Rule 18(b), each party has fourteen (14) days within which to request redaction “of any information furnished by that party: (1) that is a trade secret or commercial or financial in substance and is privileged or confidential; or (2) that includes medical files or similar files, the disclosure of which would constitute a clearly unwarranted invasion of privacy.” Vaccine Rule 18(b). Otherwise, the entire Decision will be available to the public in its current form. *Id.*

<sup>2</sup> The Vaccine Program comprises Part 2 of the National Childhood Vaccine Injury Act of 1986, Pub. L. No. 99-660, 100 Stat. 3755 (codified as amended at 42 U.S.C. §§ 300aa-10–34 (2012)) (hereinafter “Vaccine Act” or “the Act”). All subsequent references to sections of the Vaccine Act shall be to the pertinent subparagraph of 42 U.S.C. § 300aa.

Having had the opportunity to consider the medical records, expert reports, and testimony adduced at hearing, I now deny entitlement to a damages award. This claim arises from an immense tragedy—but my sympathies for the Martins’s suffering are not a sufficient basis for a favorable decision. At bottom, the causation theory offered herein is scientifically unreliable, especially in light of the unpersuasive testimony offered by two of Petitioners’ three experts. And although it is not unreasonable for Petitioners to have speculated that a vaccine I.R.M. received two days prior to his death might have played some role in the occurrence, the medical record in this case does not preponderantly support that conclusion.

## **I. Factual Background**

I.R.M. was born full-term on September 1, 2011. Ex. 8 at 103. Before he was two years old, I.R.M. was noted to have an ear deformity (Ex. 3 at 170), and he had tubes placed in his ears due to recurrent ear infections. Ex. 4 at 201, 206-7; Ex. 3 at 153. The record establishes no other notable health concerns. For his early years of life, I.R.M. received his usual vaccines on schedule without notable complications.

On September 24, 2014, at approximately 4:00pm, I.R.M. (now three years old) received a dose of Flumist (the quadrivalent live attenuated influenza vaccine (“LAIV”))<sup>3</sup> intranasally (the manner of this particular vaccine’s administration) at a well-child visit to his pediatrician. Ex. 3 at 234–39. The records reveal no evidence of any immediate or transient reaction of the kind often associated with vaccination, such as fever or lethargy. Indeed, as Petitioners have admitted, I.R.M. was active and playing on the first day following vaccination—September 25, 2014 – with no indication of a concerning reaction. Ex. 73 (Affidavit of Lindsey Martin, dated March 25, 2019) at 1-2. By the morning of September 26, 2014, however—now almost 40 hours post-vaccination—I.R.M. seemed tired in the recollection of Mrs. Martin, who reports unusual difficulties in waking him. Pet. at 1; Ex. 9; Ex. 73 at 2.

Mrs. Martin took I.R.M. to his babysitter’s home later that morning. Ex. 78 at 1 (declaration of Karla Sue Hubacher). Although the babysitter recalls that morning passing “unremarkably,” she asserted in statements prepared closer to the 2019 trial date that I.R.M. seemed tired and was reluctant to eat; her contemporaneous statement to police officers noted no such concerns. *Id.*; Ex. 55 at 15. She also has maintained in more recent statements that I.R.M. specifically informed her that he did not feel well, and she therefore opted to have him nap, still assuming that he was simply tired. Ex. 78 at 1–2.

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<sup>3</sup> “LAIV; a live, attenuated, cold-adapted trivalent vaccine containing temperature-sensitive type A and B strains of influenza virus that can replicate in the nasal passages but not in the lower respiratory tract; administered intranasally for immunization against influenza in persons ages 2 through 49 years.” *Influenza Virus Vaccine Dorland’s Illustrated Medical Dictionary Online*, <https://www.dorlandsonline.com/dorland/definition?id=116536> (last visited May 5, 2020) (Dorland’s Online).

At approximately 1:30 p.m., I.R.M. was put down for a nap. Ex. 4(2) at 125; Ex. 55 at 15. The babysitter informed the initial police responders that she had checked on I.R.M. during his nap and had noticed nothing of concern. However, when Mrs. Martin later came to pick I.R.M. up around 4:00 p.m., she discovered him face down in vomit, his nose and ears discolored, and his jaw stiff, making CPR difficult. Ex. 55 at 15; Ex. 73 at 3. Paramedics were contacted and transported I.R.M. to the Riverside Hospital emergency room in Columbus, Ohio, where resuscitation attempts continued in the emergency room. Tragically, all such efforts proved unsuccessful, and I.R.M. was pronounced dead at 5:05 p.m., on September 26, 2014. Ex. 4(2) at 125–26; Ex. 55 at 15.

Autopsy efforts thereafter to ascertain the cause of I.R.M.’s death were complicated by the fact that key organs, including the heart, had been harvested for donation, inhibiting direct dissection or visualization of the heart grossly or microscopically, to rule out cardiac infections or cardiomyopathies. Ex. 4(2) at 135; Ex. C at 6. The autopsy report, prepared by Dr. Kent Harshbarger, a forensic pathologist for Franklin County, Ohio, and based on examination performed September 28, 2014, officially states that the cause of death was undetermined, with no congenital abnormalities or signs of trauma noted. Ex. 5 at 142. The pathological findings at autopsy for I.R.M included mild to moderate pulmonary edema and congestion, mild to moderate cerebral edema and congestion, equivocal focal acute hypoxia-ischemia changes in the hippocampus, and hippocampal malformation. Ex. C at 6 (citing Ex. 5). Genetic testing could not rule out a cardiac channelopathy. Ex. 6.

## **II. Expert Reports and Hearing Testimony**

### *A. Petitioner’s Experts*

1. Dr. Douglas Miller – Dr. Miller, a neuropathologist, testified at hearing and filed a single expert report. Report, dated April 4, 2018, filed as Ex. 70 (ECF No. 60-1) (“Miller Rep.”). He offered the opinion that the Flumist vaccine likely caused I.R.M.’s death, based on his contention that I.R.M. possessed a previously-undiagnosed structural brain abnormality that made him susceptible to seizures in sleep capable of causing death. Tr. at 35–36.

Dr. Miller studied biology in college and graduated from Williams College in 1974 with highest honors. Miller CV, filed on April 9, 2018 as Ex. 71 (ECF No. 60-2). He then earned his M.D. at the University of Miami School of Medicine in 1978. *Id.* at 1. Two years later he obtained his Ph.D. in physiology and biophysics from the University of Miami. *Id.* After his formal education he began his residency training at the Massachusetts General Hospital, where he was a anatomic pathology resident from 1980 to 1982. *Id.*; Tr. at 5. Then he served as a neuropathology resident from 1982 to 1984. Tr. at 5. Dr. Miller also serves on the editorial boards of three neurology or neuropathology journals. Tr. at 10. Dr. Miller is not an immunologist. *Id.* at 36.

Dr. Miller is currently a clinical professor at the University of Missouri School of Medicine, and (since the fall of 2018) has been interim chair for the Department of Pathology and

Anatomical Sciences, where he performs different teaching duties. Tr. at 6–8. He identified himself as the sole neuropathologist in Central Missouri, resulting in him being called on to assist medical examiners in autopsies (sometimes in criminal contexts) where neuropathologic issues are raised. *Id.* at 7. In such circumstances, he will commonly examine brain or other central nervous system tissues to evaluate “unexpected findings.” *Id.* at 8–9.

Dr. Miller first discussed his evaluation of the coroner’s report for I.R.M. He deemed the “most important thing” derived from the report the fact that it did not specify “an anatomic cause of death,” meaning (as the examiner concluded) I.R.M.’s death fell generally into the category of sudden unexpected death of a child, or “SUDC.” Tr. at 12, 13. He noted that the medical examiner observed no presence of an infectious process in the lungs, although they were slightly heavy and revealed some congestion and edema. *Id.* at 12.<sup>4</sup> He considered I.R.M.’s brain weight, however, to be remarkable for a three-year-old, adding that the brain itself showed the existence of cerebral edema or swelling. *Id.* at 12–13, 38–39. He also noted that genetic testing performed by the examiner to look for evidence of congenital cardiac arrhythmias was negative. *Id.* at 13. Overall, Dr. Miller did not contest the examiner report’s findings, and observed that his interpretation of them was little different from the observations of Respondent’s pathologist expert, Dr. Harris. *Id.* at 24, 64–65.

Beyond the report, Dr. Miller evaluated slides of brain tissue samples taken from I.R.M. He noted that one slide revealed a “piece of temporal lobe with hippocampus,” part of the brain that he deemed critical to memory formation. Miller Rep. at 4; Tr. at 14. He particularly pointed out the “dentate gyrus” section of the hippocampus tissue sample (which, he explained, resembles a sweeping animal horn). *Id.* at 15. This section is comprised of a curved “line of small granular neurons,” and acts as a gate to regulate impulses from the nervous system coming into the hippocampus (a brain zone in which seizure activity can be generated). *Id.* at 14, 18. The neurons of which the dentate gyrus is constituted are particularly sensitive to lack of oxygen (hypoxia) or reduced blood supply (ischemia). *Id.* at 14–16.

Normally the dentate gyrus would be expected to form a thin and narrow band of tightly-packed cells, but the dentate gyrus evident from the slide in question appeared “anatomically abnormal,” something Dr. Miller proposed I.R.M. had likely been born with (and thus pre-dated vaccination). Tr. at 16, 41. Medical studies, however, have associated abnormal development of the dentate gyrus with not only seizure propensity (since it was often seen in patients diagnosed with epilepsy), but also have proposed that this kind of abnormality might explain sudden infant death syndrome (“SIDS”) as the product of single nocturnal seizure, although SIDS has been more closely associated with other risk factors (in particular sleeping in a prone position). *Id.* at 17–18, 41, 43; H. Kinney et al., *Sudden Death, Febrile Seizures, and Hippocampal and Temporal Lobe*

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<sup>4</sup> Dr. Miller did allow for the possibility that I.R.M. might have had asthma based upon his autopsy lung presentation. Tr. at 38.

*Maldevelopment in Toddlers: A New Entity*, 12 *Pediatr. Dev. Pathol.*, 6:455–63 (2009), filed as Ex. 64 (ECF No. 46-1) (“Kinney I”). Dr. Hannah Kinney, a Harvard Medical School pathologist, is the primary author of Kinney I, and she has performed extensive research into the possible causes of SIDS and SUDC.

Such a sleep seizure might result in a “fatal event” characterized by a cessation of breathing and brain edema. Tr. at 18. Dr. Miller deemed it “well settled” that children with this particular brain abnormality possess a risk factor for unexplained death, noting that they are overrepresented amongst those with diagnosed epilepsy who die (an occurrence termed sudden unexplained death in epilepsy, or “SUDEP”), as well as in SIDS or SUDC. Tr. at 19–20, 41, 62, 293.<sup>5</sup> He later admitted, however, that Kinney I expressly did *not* state that this hippocampal anomaly was also associated with SIDS—and although he claimed familiarity with “subsequent studies” since Kinney I’s publication in 2009 making this link, he did not file any in this matter.<sup>6</sup> *Id.* at 43; Kinney I at 8 (“[a]lthough prone sleep position is associated with SIDS, febrile seizures and hippocampal abnormalities are not”). He also noted that because the presence of the abnormality could only be detected on autopsy, its exact causal role in seizure or sudden unexplained death was ultimately unknown. *Id.* at 43, 44 (“[y]ou can’t say that the seizure was generated by the abnormality”).

Another component of Dr. Miller’s causation opinion was rooted in the interplay of the concept of “seizure threshold” with brain abnormalities akin to what I.R.M. is alleged to have possessed. The neurons making up the dentate gyrus are pyramidal, or triangular, in structure, and evidence of their cell death (whether due to hypoxic or ischemic insult) can easily be discerned under magnification given the idiosyncratic form of these neurons. Tr. at 20–21. A child possessing the abnormality alleged herein could be characterized as having a lower threshold to seizure, and might even have had subclinical seizures in the past. *Id.* at 22. Thus, evidence of some prior subclinical seizures would support the contention that a later seizure explained sudden death. *Id.* at 24. He saw some such evidence from I.R.M.’s brain tissue slides (although he admitted the evidence was at best “suggestive” of prior seizures). *Id.* at 23.

In a subsequent discussion of some of the SUDC-oriented literature filed in this case (and some items that were only filed after), including Kinney I, Dr. Miller acknowledged that these articles largely did not address “vaccination or infection to any significant degree at all” as a possible risk factor. Tr. at 293, 299–301. But he emphasized Kinney I’s embrace of the concept

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<sup>5</sup> Dr. Miller claimed to be “in the middle of” preparing his own paper based on a survey of over 1,000 child autopsies that he represented further supported his contention of the association between this brain abnormality and unexplained death, although it was not filed in this case (and may not yet have been published). Tr. at 19.

<sup>6</sup> After the hearing, Petitioners filed an additional article co-authored by Dr. Kinney addressing SUDC, but which was created two years *before* the 2009 Kinney article discussed by Dr. Miller at hearing. *See* H. Kinney et al., *Sudden Death in Toddlers Associated with Developmental Abnormalities of the Hippocampus: A Report of Five Cases*, 10 *Pediatr. Dev. Pathol.* 208-23 (2007), filed as Ex. 80 (ECF No. 76-2). This article is also identical to Petitioners’ earlier, timely-filed Ex. 31.

that the dentate gyrus abnormality (which serves as “a gate for electrical impulses in the hippocampus”) results in a lower seizure threshold—meaning, in turn (in Dr. Miller’s estimation) that “anything else” (such as the early phases of sleep) could help increase the risk of seizure. *Id.* at 294–95, 301–02. Dr. Miller admitted, however, that such literature at most only *associated* various factors with sudden death—the articles did not state that such a factor *caused* a lowering of the seizure threshold. *Id.* at 302. In addition, some of these articles spoke either of febrile seizure and SUDC in the presence of the hippocampal abnormality, or acknowledged that a significant percentage of SUDC cases (a third) did not involve hippocampal abnormalities. *Id.* at 304.

On the slides containing I.R.M.’s brain tissue samples, Dr. Miller saw evidence of both existing and prior seizures. He pointed out “scattered pyramidal neurons” from one of I.R.M.’s slides that appeared to him to have suffered some kind of hypoxic/ischemic injury. Miller Rep. at 5; Tr. at 22. Such processes would have preceded I.R.M.’s death. *Id.* at 61–62. Dr. Miller even observed something that could be a “scar” reflecting prior neuron death (and thus evidencing an earlier, subclinical seizure), although he was somewhat equivocal in so asserting. Tr. at 22 (previous seizure “could” result in the finding Dr. Miller observed). Dr. Miller also proposed that one of the slides he reviewed (prepared from sample tissue at his request for this case) revealed “Chaslin gliosis,” or the abnormal presence of astrocytes,<sup>7</sup> that have in the past been considered “suggestive” of epilepsy or prior seizures—although Dr. Miller readily admitted that this evidence was “not entirely specific,” and did not otherwise prove that I.R.M. had in fact previously experienced seizures. Miller Rep. at 4; Tr. at 22–24.

Dr. Miller then turned to the more overt evidence derived from the factual circumstances of I.R.M.’s death. Noting that I.R.M. died after being put down for a nap, and that he was found with vomit around his head, Dr. Miller deemed it likely that I.R.M. suffered “the kind of event that Dr. Kinney and her colleagues have been describing in association with his hippocampal abnormality”—a seizure event in sleep. Tr. at 24. A reasonable explanation for *why* I.R.M. suffered a seizure at this time was, in Dr. Miller’s view, I.R.M.’s receipt of the Flumist vaccine a few days prior. *Id.* at 25. Vaccination of this kind would, he maintained, cause the production of “a variety of inflammatory cytokines” that could thereafter cross the blood-brain barrier, enter the brain, and (among other things) help lower the seizure threshold, contributing to a predisposition to seizure (as Dr. Miller surmised was the case with I.R.M., given the evidence of his hippocampal abnormality). *Id.* at 25–26.

Those cytokines would include “neuromodulators or synaptic modulators” that could impact “the electrical activity of the brain,” thereby causing a lowered seizure threshold in a different way. Tr. at 34, 295. Dr. Miller did not, however, identify a specific cytokine that might be to blame for this aberrant response (and disclaimed the expertise necessary to do so). *Id.* at 54–

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<sup>7</sup> Chaslin gliosis is “[a] condensation of usually delicate horizontal glial fibers that are normally found in the cortex, immediately beneath the pial surface, thought to be a consequence of convulsions.” William Pryse-Phillips, *Companion to Clinical Neurology* 180 (2003).

55. He also proposed that sleep itself was probably a contributing factor, noting that the connection between the relevant hippocampal abnormality and sudden child death typically observed death occurring while sleeping. *Id.* at 27–28. He deemed the risk heightened during the non-rapid-eye-movement (“REM”) sleep phase. *Id.* at 44–45.

Importantly, Dr. Miller admitted that he could not with certainty diagnose a pre-death seizure as having occurred in I.R.M.’s case, given that a seizure is an “electrical event,” the evidence of which would not be present at the time a pathologist reviewed brain tissue samples. Tr. at 31. He also agreed it could not be determined from the medical examiner report precisely when such possible prior seizures might have occurred (although he speculated it could be virtually any time in a given day, even if most likely during sleep) or what might have triggered them. *Id.* at 48–49, 63. But he felt nevertheless the brain abnormalities he observed were “hints” that seizure activity was possible for I.R.M., and might have previously occurred. *Id.* at 31–32.

Regarding when I.R.M.’s alleged vaccine-caused seizure occurred, Dr. Miller noted that prior to being placed down for a nap, there was no outward evidence that I.R.M. was experiencing any kind of encephalopathic process. Tr. at 26. But the fact that I.R.M. was found to be somewhat stiff later that afternoon suggested that his death occurred close in time to the nap’s start (or “a couple of hours before he was found”), as it would take time for rigor mortis to occur, and I.R.M. was more likely in an early sleep cycle in which a seizure might occur. *Id.* at 27–29, 32. The subsequent death process, however, would not be immediate, but would have occurred over several minutes, as the brain was deprived of oxygen. *Id.* at 29. The evidence of I.R.M.’s swollen brain did not rebut this view, Dr. Miller asserted, noting that a child’s brain could swell rapidly, and the edema evident was not otherwise inconsistent with sudden death in sleep. *Id.* at 30.

Dr. Miller also proposed that the timeframe from vaccination to I.R.M.’s death was medically acceptable. Although he acknowledged that “there’s a fairly rapid time course for some of these proinflammatory cytokines to have blood levels that go up and then come back down,” that would “not necessarily reflect what’s going on in the brain,” such that elevated levels of cytokines could continue to exist in the brain even while levels in the blood were declining. Tr. at 35. He added that other vaccines might have a longer timeframe from administration to onset of clinical evidence of pathology, but for “ordinary childhood vaccinations,” the risk would in his view peak between two and three days, dropping off thereafter. *Id.* at 57.<sup>8</sup> Dr. Miller noted that

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<sup>8</sup> On redirect, Dr. Miller discussed some of Respondent’s literature pertaining to the expected timeframes for the peaking of proinflammatory cytokines induced by vaccination, arguing that the better articles suggested a peak within 24 hours. Tr. at 297, 310; Y. Kashiwagi et al., *Production of Inflammatory Cytokines in Response to Diphtheria-Pertussis-Tetanus (DPT), Haemophilus Influenzae Type B (Hib), and 7-valent Pneumococcal (PCV7) Vaccines*, 10 Human Vacc. & Immunotherapeutics, 3:677–85 (2014), filed as Ex. A-9 (ECF No. 35-10) (“Kashiwagi”). Dr. Miller admitted, however, that Kashiwagi observed “roughly similar” cytokine profiles in such a timeframe before and after vaccination, whether or not a patient had experienced a fever, and also that Kashiwagi did not include the flu vaccine at all in its tests of the impact of vaccines on proinflammatory cytokine levels. *Id.* at 310. By contrast, a different article filed by Respondent was *specific* to Flumist, but showed a longer timeframe for cytokine upregulation. See M. Barria et al., *Localized Mucosal Response to Intranasal Live Attenuated Influenza Vaccine in Adults*, J. Infectious Diseases 207:115–24 (2013), filed as Ex. A-7 (ECF No. 35-8). Dr. Miller deemed the distinction in findings among

vaccine-induced cytokine upregulation was the core aspect of this component of his opinion—as well as the fact that he did not have a background in immunologic matters, and did not otherwise include this part of his opinion offered at trial in his original written report. *Id.* at 36–37, 50, 54.

There were evidentiary limitations, however, to this sub-element of Dr. Miller’s opinion. Dr. Miller admitted that he could not recall if Kinney I addressed the theorized role of cytokines in unexplained infant/child death from seizure (Tr. at 45), although he purported that because it was understood medically that fever (which can be a response to an infectious process) is mediated by cytokines, Kinney I’s implication that fever or a febrile-induced seizure might be involved in an unexplained death would mean that “cytokine actions” were in turn likely causal. *Id.* at 47. He otherwise agreed that those articles he had filed did not support this part of Petitioner’s case. *Id.* at 46-47.

Dr. Miller also acknowledged that not all cytokines were proinflammatory, yet it was *that* kind of cytokine in particular that was associated with a lowered seizure threshold. Tr. at 47. He noted that there was no way to determine from the evidence available the precise nature of the theorized aberrant cytokine response. *Id.* at 50. At most, the day prior to his death (as well as the morning of), I.R.M. was reported to be “unusually lethargic,”<sup>9</sup> which could be interpreted as evidence of “something wrong with his brain,” which would in turn likely have been cytokine-mediated. *Id.* at 58–59. And he could not reference any literature filed in this case supporting the contention that the Flumist version of the flu vaccine could cause seizures (although he claimed familiarity with unfilled literature showing “some increase in neurological manifestations” for both children and adults after receipt of this form of the vaccine). *Id.* at 53. Indeed, he could point to no “red flag” evidence from the autopsy report connecting the vaccination to I.R.M.’s death, other than the fact of the vaccination itself plus the lack of evidence of any other potentially explanatory factor. *Id.* at 57–58.

One issue coloring the entirety of Dr. Miller’s testimony was the question of whether opinions he has offered in prior Vaccine Program cases (along with existing scientific and medical literature relevant therein) involving SIDS had any bearing herein. On the one hand, Dr. Miller was careful to state that the present claim is (from a literal standpoint) “not a SIDS case,” given that I.R.M. was three when he received Flumist, whereas the SIDS classification applies only to infants who die before the age of one. Kinney at 2; Tr. at 42. The theory that a vaccine could cause SIDS also is based on the notion that “medullary abnormalities” present in infant brains, when combined with other factors, could precipitate unexplained death, factors not bearing on the death of a three-year-old (whose brain development is more advanced). *Id.* at 51. It was for this reason,

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these items of literature meaningless, however, because “vaccines work by stimulating mechanisms that are common across multiple types of antigens,” and hence in his view Kashiwagi’s findings still had weight. Tr. at 310–11.

<sup>9</sup> Dr. Miller also noted that the Flumist vaccine’s package insert expressly predicts lethargy as a possible vaccine response, adding that this response would likely be cytokine-mediated. Tr. at 59–60. It does not appear in this case, however, that the package insert was ever filed to substantiate this assertion.

Dr. Miller explained, that he mostly did not file literature relevant to SIDS that would have supported certain aspects of his opinion herein. *Id.* at 42–43, 47.

At the same time, however, Dr. Miller admitted there were “similarities” between the kind of causation theory he has offered numerous times in SIDS cases and the present matter. In particular, he acknowledged that he had previously opined about the role of cytokines in causing SIDS by impacting brain structures, but that such contentions had been generally rejected (with the exception of one case that, at least as of the time of his testimony, was on appeal).<sup>10</sup> *Tr.* at 52.

On redirect, Dr. Miller attempted to bulwark Petitioners’ contentions about cytokines crossing the blood-brain barrier (a topic that is facially outside of his primary area of expertise). He argued that “multiple papers” supported the concept, and attempted to discuss the biological mechanisms by which this could occur. *Tr.* at 296. Here, he maintained, the cytokines would be actively transported via receptors in the blood-brain barrier’s epithelial cells and/or brain blood vessel capillaries, meaning that a high concentration of cytokines in the blood was not relevant to whether transportation was occurring. *Id.* at 296–97. He added on redirect that he felt that any time an “inflammatory process” (whether due to infection or vaccination) caused an elevation of cytokines like IL-1 $\beta$  in the blood, “some level” of them would also get into the brain (although he provided no substantiation for this opinion, which unquestionably falls outside of Dr. Miller’s expertise). *Id.* at 308.

2. Dr. Marcel Kinsbourne – Dr. Kinsbourne, a pediatric neurologist by training, prepared two reports in this case and testified at hearing. *Tr.* at 65–120; Report, dated December 17, 2015, filed as Ex. 10 (ECF No. 12-2) (“Kinsbourne Rep.”); Report, dated April 28, 2017, filed as Ex. 56 (ECF No. 44-1) (“Kinsbourne Supp. Rep.”). He opined that the Flumist vaccine triggered an immune-caused reaction in I.R.M., lowering his seizure threshold and resulting in his subsequent death. *Tr.* at 69–70.

Dr. Kinsbourne is board certified in pediatrics. Kinsbourne CV at 1–2, filed on December 23, 2015 as Ex. 11 (ECF No. 12-3) Ex. 44 (ECF No. 81-1) (“Kinsbourne CV”). He received his medical degree in England, and he has been licensed to practice medicine in North Carolina since 1967. *Id.* From 1967 to 1974, Dr. Kinsbourne served as an associate professor in pediatrics and neurology and a senior research associate at Duke University Medical Center before holding a series of academic positions, including professorships in pediatrics, neurology, and psychology. *Id.* His clinical experience includes serving as a senior staff physician in Ontario from 1974 to 1980, and a clinical associate in neurology at Massachusetts General Hospital from 1981 to 1991, although (as noted in other cases) many years have passed since he regularly saw patients. He is

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<sup>10</sup> Dr. Miller was likely recalling the case *Boatmon v. Sec’y of Health & Human Servs.*, 941 F.3d 1351 (Fed. Cir. 2019). The special master’s decision in that SIDS case accepting Dr. Miller’s causation theory was overturned by the Court of Federal Claims, and the Federal Circuit upheld that determination - although the Federal Circuit had not yet issued its decision as of the date of Dr. Miller’s testimony in this matter.

on the editorial board of several journals that deal with the brain, such as *Brain and Cognition* and *Brain Research*. Kinsbourne CV at 2–3.

Cross-examination of Dr. Kinsbourne established a number of reasons to question the degree to which his professional qualifications and experience (especially at present) made him a good “fit” to opine on the issues in contention. Specifically, he admitted (a) having no hospital-based clinical practice for approximately 25 years, (b) that he only occasionally sees patients on referral, (c) that he had not treated a seizure or evaluated its cause in that entire period, and (d) that his focus since ceasing to regularly see patients had been on teaching (mainly as a professor of psychology teaching neuroscience to graduate psychology students), although he argued that he nevertheless frequently considered issues pertaining to neurology and the brain. Tr. at 67, 87–90. He similarly acknowledged that he lacked training and expertise in immunologic matters generally, and the study of cytokines specifically, deferring on such issues to Petitioner’s third expert, Dr. Levin. *Id.* at 91–92.

Dr. Kinsbourne began his testimony by noting that I.R.M. had generally been healthy before receiving the Flumist vaccine. Tr. at 70. However, by the second day after vaccination, he seemed lethargic to his caregivers—the kind of reaction well understood, Dr. Kinsbourne maintained, to be vaccine caused, and more specifically cytokine-mediated. *Id.* at 71–72.<sup>11</sup> To explain how this could occur, Dr. Kinsbourne proposed that an innate (and hence mostly immediate and nonspecific) reaction to vaccination would generate proinflammatory cytokines that would “enter the brain” and specifically impact the hypothalamus. *Id.* at 71–73.

Dr. Kinsbourne next reviewed (and largely reiterated) Dr. Miller’s testimony, endorsing its logic but endeavoring to add some detail to the theory presented earlier at hearing. *See generally* Tr. at 73–77. He thus agreed with Dr. Miller that the hippocampal region of the brain had significance with respect to seizures, and that a child with a “dentate abnormality” would be especially susceptible to seizure activity due to a lower threshold for seizure, although that susceptibility was distinguishable from a seizure trigger. *Id.* at 74–75, 103. He also briefly discussed Kinney I in the context of such brain abnormalities, noting that even though the article did not specifically identify vaccines as a trigger for seizure under such circumstances, it *did* mention infection, and “one could conceive of a vaccination as being a mild controlled infection.” *Id.* at 76.

The opinions expressed in Dr. Kinsbourne’s two reports are inconsistent, a fact he readily acknowledged at hearing. In his first report, he admitted, he was “basically deferring to Dr. Levin” that brain swelling had caused I.R.M.’s death. Tr. at 98. However, he subsequently altered his

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<sup>11</sup> Dr. Kinsbourne discounted the possibility that this lethargy could have reflected a postictal state, i.e. recovery from an undiagnosed seizure the night before I.R.M.’s death. Tr. at 315–16. The description of I.R.M.’s status from the record and the recollection of Mrs. Martin only suggested that I.R.M. did not feel well, not that he “was in the lower level of consciousness” that would reflect a true postictal state. *Id.* at 316–17.

opinion in conformity with Dr. Miller's views about the purported brain abnormality observed in autopsy and its likely association with seizure susceptibility. *Id.* at 92, 98, 101–02. He did note that the autopsy revealed brain swelling, but attributed it to the seizure (rather than being the cause of it, as he had previously opined). *Id.* at 99; Kinsbourne Rep. at 4 (“[t]he cytokine storm caused [I.R.M.’s] brain cells to swell and rendered cerebral blood vessels permeable, permitting edema to accumulate,” and thereafter resulted in his death).<sup>12</sup> He affirmatively stated that his most up-to-date opinion in this case was to be found in his second report. Tr. at 119.

In particular, Dr. Kinsbourne maintained, a vaccine like Flumist would contain some live viral antigens comparable to a natural infection, which could have the same triggering effect, and likely affected I.R.M. in the timeframe experienced. Tr. at 76–77, 112. As a LAIV, Flumist is administered by spray directly into the mucosal areas of the nose, and formulated to induce a systemic response akin to what is “observed after natural infection,” but without the degree of viral replication in the upper or lower respiratory areas. *Id.* at 77–78; R. Cox et al., *Influenza Virus: Immunity and Vaccination Strategies. Comparison of the Immune Response to Inactivated and Live, Attenuated Influenza Vaccines*, 59 Scand. J. Imm. 1-15 (2004), filed as Ex. 19 (ECF No. 13-1) (“Cox”). Dr. Kinsbourne deemed Flumist particularly useful for young children, given its method of administration, and added that it would result in the production of proinflammatory cytokines that he maintained would be “actively transported” into the brain, although he did not specify how this would actually occur. *Id.* at 79.

On cross examination, Dr. Kinsbourne admitted he could not explain why prior vaccinations I.R.M. had received had not similarly triggered seizure. Tr. at 104. He could only observe that it was common in vaccine injury cases for a child to have no prior reaction before a more serious occurrence, noting that in such cases “other circumstances in place” were probably contributing to the vaccine’s impact. *Id.* Any prior vaccines I.R.M. received had likely also caused a cytokine response akin to what he opined had occurred here, but medical science simply could not explain why those occasions did not produce the fatal outcome at issue. *Id.* at 105.

To bulwark his contentions about the capacity of the flu vaccine generally to cause injury, Dr. Kinsbourne referenced some literature discussing encephalopathies in children believed to be associated with a wild flu virus strain. Tr. at 80–82; T. Togashi et al., *Influenza-Associated Acute Encephalopathy in Japanese Children in 1994-2002*, 103 Virus Res. 75–78 (2004), filed as Ex. 51 (ECF No. 15-8) (“Togashi”). In Togashi, a particular flu viral strain (also included in the version of Flumist received by I.R.M.) was detected in the cerebrospinal fluid of a group of Japanese children suffering from an acute encephalopathy. Tr. at 81; Togashi at 77. A large percentage of the studied sample also displayed increased levels of several different proinflammatory cytokines (the existence of which Dr. Kinsbourne attributed to an immune response), and Togashi speculates

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<sup>12</sup> Dr. Kinsbourne also admitted that an element of the opinion in his first report that had been carried over to the second, despite the change in overall focus—that “cytokines upregulate glutamate production” sufficient to induce brain edema—was likely inaccurate (and certainly no longer relevant given his reliance on Dr. Miller’s opinion). Tr. at 100 (“I’m not sure that I was right about that”), 101.

that the measured cytokines might have played some pathologic role in the disease process, perhaps by weakening the blood-brain barrier. Togashi at 78. But Dr. Kinsbourne admitted that he did not contend that I.R.M. had suffered an encephalopathy, and Togashi's authors say nothing about vaccines being potentially causal of any injury process associated with encephalopathy—in fact, they reached the *opposite* conclusion. *Id.* (“[t]he best way to avoid this severe complication is no doubt prevention by influenza vaccination”); Tr. at 107. Dr. Kinsbourne also claimed that Cox observed increased levels of similar cytokines after vaccination, although that article does not squarely address post-vaccination cytokine levels, focusing instead on antibody levels. Cox at 5–7.

These cytokines, Dr. Kinsbourne opined, would “make neurons more excitable,” thereby exceeding certain levels maintained in the brain. Tr. at 85. As the ratio between inhibiting factors and exciting factors (here, proinflammatory cytokines allegedly induced by vaccination) became unbalanced, the seizure threshold would be reduced, thereby increasing the risk of seizure. *Id.* at 85–86, 108. Thus (and consistent with Dr. Miller's testimony), I.R.M.'s purported brain malformity already rendered him susceptible to seizure, but the addition of a vaccine that encouraged lowering of the threshold only worsened conditions. *Id.* at 86.

Dr. Kinsbourne was unsure as to how much excess cytokine dysregulation *per se* had caused I.R.M.'s proposed seizure, versus a “modest amount” of cytokines being sufficient to precipitate a lowered threshold in a child with a susceptibility due to brain malformity, as alleged to be the case here. Tr. at 99–100. He similarly could not identify the specific cytokines likely most responsible herein for allegedly helping trigger a seizure in I.R.M. At most, he noted that literature associated IL-1 $\beta$  with seizure activity, although he had “no idea” how much of the cytokine would be required to induce seizure. *Id.* at 106. He also was unable to opine as to how long the brain would have to be exposed to cytokines to have resulted in the purported seizure in question, speculating that perhaps 10 to 15 minutes would be sufficient. *Id.* at 116–17. He did, however, maintain that I.R.M.'s purported lethargy the morning prior to his death could establish the existence of elevated cytokine levels. *Id.* at 118. Ultimately, Dr. Kinsbourne acknowledged that the medical science relevant to how cytokines might encourage seizures was beyond his ken (although he believed there was support for the contention), and deferred to Dr. Levin on the subject. *Id.* at 96–97, 117.

There were other acknowledged deficiencies with Dr. Kinsbourne's aforementioned theory. First, he agreed that regardless of his personal views, it was not “well known” among the medical and scientific community that Flumist vaccine causes seizure regardless of mechanism. Tr. at 97. Indeed, he could not identify literature directly addressing the contention, although he proposed that what was known about the association between other flu vaccines and seizure was relevant as well to a different formulation of the flu vaccine, like Flumist. *Id.* He also allowed that literature such as Togashi involved wild flu virus-induced fever preceding encephalopathies, and not vaccines. *Id.* at 97–98. Here, there is no evidence that I.R.M. had a fever on the morning of his

death (and indeed beyond claims of lethargy, no evidence he experienced any reaction at all post-vaccination).

The circumstances in which I.R.M. died were significant to Dr. Kinsbourne's opinion—in particular the fact that he had been asleep. Dr. Kinsbourne distinguished between deep REM sleep, when dreaming occurs, and the lighter, initial phase (non-REM sleep). Tr. at 83. The earlier non-REM phase was the time of greatest seizure susceptibility, and hence when I.R.M. most likely experienced the alleged seizure that caused his death—a conclusion bulwarked by the extent of rigor mortis that I.R.M. displayed when he was discovered three or four hours after being placed down for a nap. *Id.* at 83-84, 114. I.R.M.'s existing seizure threshold had, Dr. Kinsbourne proposed, likely been lowered by the vaccine received two to three days earlier. *Id.* at 84–85. Of course, the record in this case reveals that I.R.M. slept *two nights* post-vaccination without incident (other than allegations of seeming lethargic upon waking) prior to the nap in question, but Dr. Kinsbourne nevertheless maintained that (to his knowledge) this was the first time I.R.M. had “gone to sleep in the period that followed an influenza vaccination.” *Id.* at 84.

On cross, Dr. Kinsbourne agreed that existing literature on SUDC did not discuss whether vaccines could trigger death. Tr. at 108–09. Yet such literature *did* observe other factors—in particular sleep position and the very fact of sleeping were linked to unexplained death, along with febrile seizures for children with hippocampal abnormalities. *Id.* at 109-11; Kinney at 5–8. He accepted the possibility that a different trigger could have caused the relevant purported seizure. *Id.* at 114–15. However, the time interval from vaccination to I.R.M.'s death was in his view acceptable enough to conclude the vaccine was the most likely trigger here. *Id.* at 116.

3. Dr. Alan Levin – Dr. Levin, a trained immunologist and pathologist, testified at hearing and prepared two reports as well. Tr. at 121–67; Report, dated December 17, 2015, filed as Ex. 12 (ECF No. 12-4) (“Levin Rep.”); Report, dated April 27, 2017, filed as Ex. 57 (ECF No. 44-2). He proposed that I.R.M.'s receipt of Flumist was associated with his death, although (like Dr. Kinsbourne) he revised his opinion somewhat after Dr. Miller weighed in with a pathologist's reading of the medical examiner's report.

Dr. Levin has a master's degree in biochemistry and received his medical degree from the University of Illinois in 1964. Levin CV at 1, filed on Dec. 23, 2015 as Ex 13P (ECF No. 12-5). Dr. Levin also received a juris doctor from Golden Gate University in San Francisco in August 1995 and currently practices law. *Id.* at 1–2. He is board certified in allergy, immunology, and clinical pathology. *Id.*; Tr. at 122. His CV lists numerous publications and states these publications are primarily in the subjects of immunology, immunopathology, cancer biology and treatments. *See* Levin CV at 4–5.

Dr. Levin claimed as well to have done research on immunologic issues, but “mostly for the Vaccine Court”—suggesting he deems work on petitions as an expert to be akin to research into the background medical or scientific question. Tr. at 123. He is not board certified in

neuropathology, however (unlike Drs. Miller and Harris), and deferred to Dr. Miller on issues in this case relevant to pathology (while maintaining he did have some up-to-date expertise with pathologic issues). *Id.* at 143–44.

As with Dr. Kinsbourne, Respondent devoted some cross-examination time to highlighting issues with Dr. Levin’s expert qualifications. In particular, Dr. Levin graduated from law school 25 years ago, and appears largely since that time to have been a practicing attorney rather than immunologist or pathologist (although the clients he had represented often have brought claims that impinge on the kinds of medical and scientific issues litigated in the Vaccine Program). Tr. at 138–39. He is not the primary physician for any patients today, even though he does see a few patients every month, and (as noted above) has kept up to date his medical licenses. *Id.* at 140–41. He also has not published any medical academic articles for over 20 years. *Id.* at 141.

Dr. Levin began his testimony with a general discussion of the immune system. He objected to separating it into innate versus adaptive branches, maintaining instead that it is better thought of as an overall system of “growth and differentiation,” through which animals have developed means of responding to pathogens in the environment. Tr. at 124–25, 136. Traditionally, the innate branch reflects the “soldiers at the gate”—cells that act in immediate response to pathogenic patterns with some degree of recognition. *Id.* at 125–26. The adaptive branch, by contrast, is comprised of cells that can respond to invading pathogen “tertiary structure” in a more specific way, but which takes longer to be effective. *Id.* at 126. Vaccines function by immediately provoking an inflammatory response in which cytokines generated in response to the vaccine in turn communicate with, and thereby activate, other immunologic cells (including anti-inflammatory cytokines that work later to “turn off the immune response”). *Id.* at 126–27.

In the kind of aberrant response to a vaccination alleged to have occurred in this case, Dr. Levin reasoned, the driver of the disease process is the body’s immune response, which involves inflammation. Tr. at 127, 129. Citing Cox, Dr. Levin observed that Flumist (as a LAIV) was formulated to cause a “very vigorous protective response” that would be more effective than the more-typical subcutaneous injection, because the LAIV would introduce a small part of a live virus into the nose, thereby ensuring a systemic response comparable to what would be experienced after a wild virus infection (albeit on a more limited scale). *Id.* at 129–32. The fact that the relevant formulation of the vaccine likely contained the H3N1 wild virus strain was also significant, and he deemed it a “superantigen” that would provoke an even more vigorous immune response. *Id.* at 152–53.<sup>13</sup>

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<sup>13</sup> Dr. Levin claimed that a number of papers supported this contention, although some of them were not referenced in his report (or were only filed on the eve of hearing). Tr. at 153; W. Fischer et al., *Live Attenuated Influenza Vaccine Strains Elicits a Greater Innate Immune Response Than Antigenically-Matched Seasonal Influenza Viruses During Infection of Human Nasal Epithelial Cell Cultures*, 32 Vaccine 15:1761–67 (2014), filed as Ex. 77 (ECF No. 71-1) (“Fischer”). But although Fischer (a study mainly seeking to compare the viral replication allowed by a LAIV versus a wild type flu virus comparable to the LAIV’s viral components) did establish some of the immunologic benefits of a LAIV, it says nothing about the allegedly *pathologic* effect of the cytokines such a vaccine would upregulate (which

Because of the vaccine's strong immune system-stimulating properties, Dr. Levin reasoned, I.R.M. likely experienced "systemic" inflammation, outside of the central nervous system, from receipt of the Flumist vaccine. Tr. at 156. He did not characterize this immune response as a "cytokine storm," however (contrary to Dr. Kinsbourne's initial report, filed simultaneously with Dr. Levin's first report). *Id.* at 157; *see* Kinsbourne Rep. at 4. As a result, he argued, the Flumist vaccine caused a seizure through the purported introduction of proinflammatory cytokines into the brain. Tr. at 137, 152, 161.<sup>14</sup> Notably, however, Dr. Levin's reports (which were filed prior to Dr. Miller's) make *no mention* of seizure as having played any role in I.R.M.'s death; Dr. Levin maintained at hearing that he "probably" did discuss seizure in one of the reports, but this is incorrect. *Id.* at 155; *but see* Levin Rep. at 7 ("[t]he most probable diagnosis is respiratory arrest secondary to cerebral edema"). His supplemental report is similarly focused on cerebral edema.<sup>15</sup>

Dr. Levin could not identify the amount of cytokines necessary to spark this purported reaction, but stated that in his opinion I.R.M.'s immune response had been sufficiently "dysregulated" to harm him—largely due to some innate susceptibility to an immune response (which he did not equate with the purported susceptibility to seizure that I.R.M.'s brain abnormality was alleged to have incurred). Tr. at 150, 152. To support such contentions, Dr. Levin discussed certain literature filed in this case that he maintained observed a relationship between seizure activity and certain cytokines, like IL-1 $\beta$ . Tr. at 163–64; A. Vezzani et al., *IL-1 Receptor/Toll-like Receptor Signaling in Infection, Inflammation, Stress and Neurodegeneration Couples Hyperexcitability and Seizures*, 25 *Brain, Behavior and Imm.* 1281–89 (2011), filed as Ex. 69 (ECF No. 69) ("Vezzani").

Vezzani (a review article) discusses some prior study's findings that identified certain cytokines (including IL-1 $\beta$ ) within the CNS as contributing to the pathogenesis of existing seizures, through impact of neuron excitability (as mentioned by Dr. Kinsbourne). Vezzani at 1283. However, Vezzani focuses more on the expression of such cytokines from *inside* the brain by immune cells located there, like microglia, and/or attributable to direct/existing brain infection or injury, than on the concept alleged herein—that cytokines induced by the immune system in the

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did *not* include IL-1 $\beta$ ), although Dr. Levin claimed that he believed such articles did stand for that conclusion. Tr. at 154; Fischer at 6–7.

<sup>14</sup> Dr. Levin also cited other literature previously referenced by Dr. Kinsbourne, like Togashi, to underscore the point that components of Flumist had been associated with neurologic injury in the past. Tr. at 132–33. However, his opinion did not seem to turn on the proposition that any specific components of the vaccine are central to the harm it allegedly caused, as opposed to the vaccine's general capacity to provoke an inflammatory response. *Id.* at 161–62.

<sup>15</sup> Even though Dr. Levin appeared at hearing to have accepted Dr. Miller's opinion that seizure was the best explanation for I.R.M.'s death, he had trouble letting go of his initial assertion that intracranial pressure caused by brain edema was to blame for I.R.M.'s death. Tr. at 148–49, 156. In particular, he maintained the cytokines *also* impacted the separation of endothelial cells (making up the blood-brain barrier) in the brain, resulting in more edema than should normally occur. *Id.* at 166–67.

periphery would migrate into the CNS—and says nothing about the capacity of vaccines to initiate such a process. Vezzani at 1287–88 (“the evidence that innate immune responses . . . may converge on common targets to increase neuronal excitability which can be sufficient to trigger seizures . . . provides mechanistic insights to bridge the proposed causal link between *CNS infections or brain damage* and epilepsy”) (emphasis added).

Dr. Levin accepted that I.R.M. had previously received the flu vaccine without a similar reaction, but stressed the significance of the Flumist version as the special factor precipitating injury. Tr. at 150–51. He deemed significant that Flumist is administered in the nose, since this occurred in “proximity to the brain”—although he did not explain how such proximity made a pathologic reaction more likely, other than assuming that this was self-evident. *Id.* at 151, 152. Dr. Levin did emphasize that, under his theory, cytokines produced in response to the vaccine needed to reach the brain (and thus cross the blood-brain barrier) in order to have the effect alleged. *Id.* at 159. He agreed on cross-examination, however, that the presence of cytokines in the brain could also be the *result* of seizures rather than solely their cause. *Id.* at 319.

Regarding timing, Dr. Levin deemed the almost two-day period from receipt of the vaccine to I.R.M.’s death to be medically acceptable. Tr. at 134–36. Certain literature, he reported, suggested the immune response to Flumist was more robust than what a wild virus would elicit. *Id.* at 133–34. Dr. Levin opined that the Flumist immune response would occur very fast, within minutes to hours. *Id.* at 131. But even though there is no evidence that I.R.M. experienced any reaction to the vaccine before the morning of September 26th (when he seemed lethargic) -- almost 40 hours post-vaccination, and thus far longer than the immediate reaction Dr. Levin seemed to embrace -- Dr. Levin still felt that this timeframe was acceptable, arguing that the alleged malaise I.R.M. displayed could be reasonably attributable to vaccine-induced cytokines. *Id.* at 135. As he saw it, the brain malformity observed from I.R.M.’s autopsy simply did not get sufficiently “excited” to react to the additional cytokines until that morning (thus suggesting it was only then that there were enough cytokines to cause a reaction). *Id.* at 158.

On cross, Dr. Levin asserted that literature on SUDC did establish that vaccines had been implicated in children’s unexplained deaths, although he could not recall what articles precisely set forth this claim. Tr. at 160. He similarly maintained that the Flumist package insert noted the possibility of seizure as an adverse event (*Id.*)—a contention that cannot be substantiated or confirmed, since Petitioners never filed the package insert as an exhibit (despite being given the opportunity to do so post-hearing). *Id.* at 60–61.

#### *B. Respondent’s Experts*

1. Dr. Brent Harris – Dr. Harris, a pediatric neuropathologist, testified for Respondent and prepared two expert reports as well. Tr. at 170–212; Report, dated December 12, 2016, filed as Ex. C (ECF No. 38-1) (“Harris Rep.”); Report, dated April 10, 2019, filed as Ex. E (ECF No. 67-1) (“Harris Supp. Rep.”). He deemed it likely that I.R.M. had experienced a seizure

resulting in SUDC, but opined that no trigger or instigating cause, including the Flumist vaccine, could be identified. Tr. at 189.

Dr. Harris earned his B.A. in biology from Colby College in Waterville, ME, in 1986. Harris CV at 1, filed on December 19, 2016 as Ex. D (ECF No. 38-9). From 1988 to 1995 he earned his master's degree in biochemistry from Hahnemann University in Philadelphia, a Ph.D. from Georgetown University, and an M.D. from Georgetown University School of Medicine. *Id.* Afterwards, he served as an anatomic pathology intern and resident at Stanford University School of Medicine from 1995 to 1997. *Id.* He was the chief resident at Stanford University School of Medicine, Department of Anatomic Pathology from 1997 to 1998. *Id.* He completed a neuropathology fellowship at Stanford University School of Medicine from 1997 to 1999 and another fellowship in Neurobiology at Stanford University School of Medicine from 1999 to 2002. *Id.*

Currently, Dr. Harris serves as an associate professor at Georgetown University School of Medicine in its pathology and neurology departments. Tr. at 171–72. He also has an adjunct role at the VA Medical Center in D.C. and as the sole neuropathology consultant for the D.C. Medical Examiner's Office. *Id.* at 172. He reviews about thirty pediatric autopsies a year—including autopsies for unexplained deaths in children and infants—for the D.C. Medical Examiner's Office. *Id.* In the last few years he has reviewed several hundred pediatric autopsies. *Id.* Dr. Harris is board certified in anatomic pathology and neuropathology and by the National Board of Medical Examiners. Harris CV at 3. He is not an immunologist and was therefore reluctant to discuss the cytokine-generative impact of vaccines. Tr. at 201.

Dr. Harris openly acknowledged the extent to which his opinion embraced many of the same conclusions set forth in Dr. Miller's opinion. Thus, he accepted the existence of I.R.M.'s brain edema resulting in it being heavy, although he utilized a comparison chart that revealed the measured weight as not significantly heavier than what was considered normal for a three-year-old boy. Tr. at 183–84, 211. He also concurred with Dr. Miller regarding the existence of the hippocampal abnormality in the dentate gyrus region of I.R.M.'s brain, and the association of such an abnormality with seizure. *Id.* at 185, 197.

Dr. Harris similarly accepted Dr. Miller's points about the presence of gliosis (as observed in the relevant brain tissue slides) suggesting that I.R.M. had suffered prior seizure activity, although he deemed the gliosis “not an entirely specific finding,” noting that it was often seen in individuals who had previously experienced seizure but did not help identify *when* such activity had occurred. Tr. at 188–89, 196. He otherwise felt that “there really wasn't much there” to consider in the examination findings; he acknowledged the existence of heavy lungs and some proof of bronchial inflammation, but noted that the heart was removed and he never saw any analysis of it (although he expressed the view it was likely normal), or any genetic findings that might have been explanatory. *Id.* at 185–86.

Besides consideration of the medical examiner's report, Dr. Harris provided his views on SUDC. He proposed SUDC was a "more generalized term" applied to sudden deaths in young children over the age of one, where "we don't have good evidence at the time of autopsy for what the exact cause of death is," often because of the absence of a known preceding illness or explanatory incident. Tr. at 178. He agreed that Kinney I (which he also filed) supported the association between hippocampus malformations and SUDC, noting that this kind of brain malformation would act as a focus for "epileptogenic activity," by causing altered "firing patterns" for neurons in this brain region. *Id.* at 179, 186, 205.<sup>16</sup> As he explained, that region of the brain was "particularly susceptible" to interruptions in oxygen or blood flow, such that seizure activity could produce visible evidence of hypoxic or ischemic damage to this part of the hippocampus—as occurred herein. *Id.* at 190–91

Dr. Harris disputed the concept that a focal seizure would always have some trigger, opining instead that the mere existence of a brain abnormality could be enough by itself. Tr. at 181, 199. He also questioned whether Kinney I had identified a precise mechanism by which death would occur in such cases, noting that at most the article identified sleep position, plus the very fact of sleep contributing to a lower seizure threshold, as primary causal factors. Tr. at 180.<sup>17</sup> Sleep position in particular was an important factor in SUDC, Dr. Harris maintained, because sleep position impacted how an individual breathes, and compromised airways (evidenced by vomit around a deceased child) were often believed to be an initiating factor in a child's seizure. *Id.* at 182.

By contrast, other related literature also co-authored by SIDS/SUDC expert Dr. Kinney had not identified vaccination as even temporally close to an instance of SUDC, and he was not otherwise aware of any literature or medical community views associating Flumist with seizure. Tr. at 189-90; H. Kinney et al., *Sudden Death in Toddlers Associated with Developmental Abnormalities of the Hippocampus: A Report of Five Cases*, 10 *Ped. & Develop. Pathol.* 208-23 (2007), filed as Ex. 31 (ECF No. 14-1) ("Kinney II"). Kinney II observed that the primary difference between SIDS and SUDC, besides age, was the discovery of the hippocampal abnormality, with sleep position being a constant risk factor for *both* SIDS and SUDC. Kinney II at 213, 219. In addition, in three of the five individual cases discussed in Kinney II, the child displayed the existence of a prior wild virus infection. *Id.* at 220.

With respect to timing, Dr. Harris felt that it was likely I.R.M. died earlier in his nap, given the evidence of edema that subsequently developed. Tr. at 177. He also felt the extent of rigor mortis observed when I.R.M. was found corroborated a pathologic process occurring earlier that

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<sup>16</sup> Dr. Harris acknowledged he is not an epileptologist with the necessary background to go deeply into this topic—although neither were any of Petitioners' experts, despite the numerous opinions offered in this case about seizure thresholds and propensities of children with epilepsy or SUDEP. Tr. at 179.

<sup>17</sup> SUDEP also often occurs at night while asleep, and without any other identifiable trigger, bulwarking the degree to which sleep might be causal factor. Tr. at 180–81. A SUDEP diagnosis would require an epilepsy diagnosis or evidence of a prior seizure, however. *Id.* at 194.

day, although he could not pinpoint the rigor's start. *Id.* The autopsy findings (which revealed hypoxic/ischemic injury mostly isolated to the hippocampus rather than widespread in the brain) suggested to him that the seizure occurred simultaneously with such localized injuries—not that one followed the other. *Id.* at 191–92. The brain edema observed in the medical examiner's report likely occurred thereafter, in response to the hypoxic/ischemic injury, and would have happened rapidly. *Id.* at 193, 195, 203, 211.

All of the above underscored for Dr. Harris the ultimately-unexplained nature of I.R.M.'s death. The hippocampal abnormality observed in autopsy suggested that I.R.M. was likely prone to seizures (or, as Petitioners prefer to say, had a lower seizure threshold). Tr. at 197, 203. However, Dr. Harris could not on the basis of this record opine as to the precise instigating factor for seizure herein, although he did accept it likely that seizure was the immediate cause of death (as well as the possibility that *some* unidentified trigger was responsible). *Id.* at 198, 203. To some extent, he said, it was just as speculative to propose I.R.M. had seized during the nap on September 26th as to deem his death attributable to vomiting while in a prone sleep position, thereby impacting his brain oxygenation. Tr. at 193–94. Ultimately, however, Dr. Harris seemed to embrace the likelihood that I.R.M.'s seizure was attributable to airway obstruction caused by vomiting in a prone sleep position. *Id.* at 206.

One question raised in Dr. Harris's testimony was the extent to which the medical record supported the conclusion that I.R.M. might have had an epileptic condition before vaccination, or would have been likely to develop one. He noted that I.R.M. could have experienced seizures earlier in his life that simply were not recognized as such, especially given the difficulties in parents understanding the relevant clinical warnings. Tr. at 181, 187. At the same time, a child could possess the same kind of hippocampal malformation without any seizure history at all. *Id.* at 187. However, if I.R.M. had experienced a pre-vaccination seizure, that made it more likely he could have one in the future. *Id.* at 188.

Dr. Harris also commented upon other aspects of Petitioners' causation theory. He questioned whether a systemic inflammatory response to the Flumist vaccine could have triggered a seizure herein, noting that the existing autopsy report provided none of the evidence that would support that contention. Tr. at 195 (“you would expect to see changes in the skin, you would expect to see influx of eosinophils in organs, changes in the weight of the organs, and we didn't see any of those changes”). He did not deem the brain edema and associated weight gain for I.R.M.'s brain to constitute such evidence. *Id.* Ultimately he rejected the contention that an immunological trigger could have caused I.R.M.'s likely seizure, speculating that (like all humans) I.R.M. had probably experienced other immune challenges in his life but weathered them without incident. *Id.* at 199, 207.

On cross examination, Dr. Harris agreed that I.R.M.'s lethargy the morning of his death was comparable to the kind of post-vaccination malaise individuals often feel, although he could not comment on whether such malaise was still possible nearly two days after receipt of a vaccine.

Tr. at 202. He also expressed uncertainty as to whether some additional factor (beyond I.R.M.'s likely seizure propensity or the fact he was sleeping) could have further "lowered" his seizure threshold, reiterating his view that a person with the hippocampal abnormality at issue could have a seizure with no additional trigger at all. *Id.* at 204–05.

2. Dr. Christine McCusker – Dr. McCusker is a pediatric immunologist, and she prepared one report and also testified at the hearing. Tr. at 213–91; Report, dated December 12, 2016, Filed as Ex. A (ECF No. 35-1) ("McCusker Rep.").<sup>18</sup> Dr. McCusker opined that the Flumist vaccine could not have instigated a seizure in I.R.M. via an innate immune response and mediated by proinflammatory cytokines.

Dr. Christine McCusker earned a master's degree in Molecular Virology in 1988, followed by an M.D. in 1993, at McMaster University, in Hamilton, Ontario. McCusker CV at 1, filed on December 19, 2016 as Ex. B (ECF No. 37-3). She served as a pediatric resident at Montreal Children's Hospital, McGill University, from 1993 to 1996. *Id.* at 1. Then, she was then a clinical fellow in allergy and immunology at McGill University from 1996 to 1999. *Id.* at 2. Dr. McCusker is board certified in pediatrics. *Id.* She is currently the division director of pediatric allergy, immunology, and dermatology at the Montreal Children's Hospital at McGill University Health Center and is the director of the Clinical Immunology Lab. Tr. at 215. *Id.* She also conducts research on developmental immunology, vaccines and immunology, and serves on the boards of several journals. *Id.* at 215–20.

Dr. McCusker's opinion in this case was rooted in some core contentions about cytokines and the roles they play in the human immune response. Dr. McCusker stressed that cytokines—proteins released by immune system cells to perform communications tasks—were not "one thing." Tr. at 236, 238. Rather, they have many different functions, and more than 80 have been identified. *Id.* at 236–37. Cytokines are often lumped together with chemokines, but Dr. McCusker maintained that the latter perform a distinguishable function (by acting as "addressants" that instruct other cells where to go, as opposed to travelling to such other cells *themselves* to deliver information, as cytokines do). *Id.* at 236.

Because of the functional importance and power of cytokines, the immune system tightly regulates their release. Tr. at 237, 247. IL-1 $\beta$ , for example (which Dr. McCusker deemed a common cytokine), is as quickly released as it is controlled, with excess amounts inactivated after being picked up by a "decoy receptor." *Id.* at 237. As a result, Dr. McCusker maintained, cytokines act rapidly and over short distances in the body, with their dissemination and circulation ultimately limited. *Id.* at 237–38. She specifically disputed the concept that cytokines themselves regularly "travel" from a peripheral site of vaccination to places like the brain, or that they could cause edema there. *Id.* at 242, 244–45. Rather (and using the example of cytokines implicated in

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<sup>18</sup> Respondent filed a second expert report from Dr. McCusker after the hearing's conclusion, and I address its contents and admissibility below.

“sickness behavior” after vaccination), cytokines responsible for fever accomplish this most often by causing peripheral nerves at the site of vaccination to communicate signals to the brain (specifically the hypothalamus), “instructing” such CNS locations to initiate a fever in response to an infection. *Id.* at 241–43, 249. Even in the circumstances of a true “cytokine storm” (which even Dr. Levin discounted as having occurred in this case) featuring uncontrolled cytokine circulation, few cytokines would still travel into the brain. *Id.* at 248–49.

Dr. McCusker did not dispute that in rare circumstances<sup>19</sup> certain cytokines (including IL-1 $\beta$ , one of the cytokines primarily identified by Petitioners’ experts) *can* permeate the blood-brain barrier, but she deemed the process usually controlled by receptors or limited in other ways. Tr. at 251–53, 287. Cytokines are also expressed directly *within* the brain by certain cellular structures located there with the capacity to produce messenger molecules, such as microglia, but the circumstances in which this would occur vary. *Id.* at 245–46. In some cases, the cells responsible for production of such cytokines inside the brain might create them in connection with the maintenance and extension of pathways relevant to memory (and in response to nerve signaling from outside the blood-brain barrier), and hence would be “key elements in all these normal brain functions.” *Id.* at 247, 249–51. Alternatively, cytokines might be produced within the brain in *response* to CNS injury, like seizure, as part of the CNS’s immune “repair mechanism.” *Id.* at 255. But Dr. McCusker rejected Petitioners’ experts’ contention that proinflammatory cytokines regularly permeate the blood-brain barrier after vaccination as a matter of course. *Id.* at 279.<sup>20</sup>

Dr. McCusker took issue with the contention of Petitioners’ experts about the kinds of post-vaccination symptoms that might be attributed to proinflammatory cytokines. Some such symptoms might be specific to the vaccine’s method of administration; thus, in Dr. McCusker’s understanding, people receiving Flumist vaccine often reported a runny nose, consistent with the vaccine’s intranasal administration (and this is the response she would usually expect to see if excess cytokine upregulation were at issue). Tr. at 239, 269–70.<sup>21</sup> Vaccines could also, she admitted, cause fever or malaise, although to her knowledge the incidence of fever was no higher than what a placebo version of a vaccine like Flumist would be expected to induce. *Id.* at 240. In

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<sup>19</sup> Dr. McCusker also noted in her report that it appeared “massive doses” of cytokines were necessary to truly permeate the blood-brain barrier, as alleged occurred here, adding that some of the literature offered by Petitioner for this proposition also required the interaction of an endotoxin for the amounts necessary. Tr. at 256–58; McCusker Rep. at 10; Tomohisa Tanaka et al., *Lipopolysaccharide Treatment and Inoculation of Influenza A Virus Results in Influenza Virus—Associated Encephalopathy—Like Changes in Neonatal Mice*, 16 J. of NeuroVirology 125–32 (2010), filed on Feb. 26, 2016 as Ex. 48 (ECF No. 17-3).

<sup>20</sup> In addressing literature offered by Petitioners to bulwark this contention, Dr. McCusker observed that the articles in question only stood for the proposition that cell structures like microglia could promote *internally* the production of different cytokines with differing functions, all as part of the process of brain maintenance—and thus the fact that cytokines were found in the brain did not mean they had traveled there from the periphery. Tr. at 279–81.

<sup>21</sup> By contrast, Dr. McCusker disputed Dr. Levin’s argument that Flumist would generally induce fever—especially in a short timeframe. Tr. at 278–79.

fact, the post-vaccination malaise that Petitioners' experts proposed had occurred here was *not* the product of the main cytokines implicated in the inflammatory response to Flumist. *Id.* at 243. She allowed, however, that certain cytokines (not including IL-1 $\beta$ ) were upregulated for a one to two-day period after vaccination, although she disputed that the amounts in question were notably elevated. *Id.* at 243-44; Y. Kashiwagi et al., *Production of Inflammatory Cytokines in Response to Diphtheria-Pertussis-Tetanus (DPT), Haemophilus Influenzae Type B (Hib), and 7-valent Pneumococcal (PCV7) Vaccines*, 10 Human Vacc. & Immunotherapeutics, 3:677-85 (2014), filed as Ex. A-9 (ECF No. 35-10) ("Kashiwagi").

Petitioners' contentions about the possible impact of cytokines on I.R.M.'s brain malformity were also addressed by Dr. McCusker. She flatly denied that cytokines would cause brain edema, as Dr. Levin posited, noting that the communication "job" cytokines perform would not directly cause such inflammation, and emphasizing that the primary cytokines located in the brain do not serve this purpose. Tr. at 259.<sup>22</sup> She also questioned how cytokines would lower seizure threshold in a child possessing the hippocampal abnormality in question. She maintained that there was no evidence in this case that in fact sufficient amounts of cytokines *were* present in I.R.M.'s brain at any time to be harmful, but even if they had been, her understanding based on existing science was that cytokine function would be the same for brains with or without such malformation—discounting the idea that the propensity for seizure from the malformity itself would also feature a sensitivity to cytokines. *Id.* at 259-60. Indeed, if Petitioners were correct in their argument, then I.R.M. should have experienced a reaction (possibly manifesting as a seizure) earlier in his life, since he likely had the hippocampal abnormality before vaccination, and had previously had other vaccines or wild infections, all of which would similarly have upregulated cytokine production. *Id.* at 261-63.<sup>23</sup>

Dr. McCusker moved on to a discussion of the Flumist vaccine itself. She agreed that Flumist, a LAIV, would cause the innate immune system to produce some proinflammatory (meaning intended to increase the immune response) cytokines. Tr. at 287-89.<sup>24</sup> Flumist is

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<sup>22</sup> Dr. McCusker later considered some of the literature cited by Dr. Levin for this proposition. Tr. at 276-77. Some of these articles involved flu infections coupled with toxicity in the body promoted by a different organ failure, while others merely reflected the creation of cytokines in *response* to disease, rather than driving it or crossing the blood-brain barrier to cause it. *Id.*

<sup>23</sup> Although, Dr. McCusker admitted, some literature filed by Petitioner did establish the existence of certain cytokines in the brain in the post-seizure period, and that introduction of cytokines like IL-1 $\beta$  could "change the excitability of neurons," there was no evidence the same cytokine was initially *causal* of the seizure – and rather was more likely a response to initial seizure activity. Tr. at 281, 82; Vezzani at 1284 (noting "evidence that IL-1 $\beta$  is produced by activated microglia and astrocytes *in response to pro-convulsant injuries*") (emphasis added). And such literature, again, did not address the transport of cytokines, induced by vaccination or otherwise, from the periphery into the brain. *Id.* at 283.

<sup>24</sup> Dr. McCusker felt it important to take care in specifying what "pro-inflammatory" meant, since not all such proinflammatory results were the same, with some cytokines having a more targeted purpose rather than simply unleashing generalized inflammation of the kind associated with infection as Petitioners' experts seemed to maintain. Tr. at 288-90.

administered in the nose<sup>25</sup> with a goal of initially developing “local immunity” at the specific site of likely infection. Tr. at 227, 234, 285; Cox at 10. By having the vaccine elicit an initial immune response in the respiratory epithelia, the chance of blocking an infectious process at that same location (where the process would otherwise commonly begin) is increased. Tr. at 234. A vaccine administered in the arm, by contrast, might create systemic immune responses, but immune cells produced from this response would need to be “called” to the airway situs of infection to be as effective. *Id.* at 235. Dr. McCusker also noted that Flumist contains “attenuated” viral strains, meaning the viral components have been processed in such a way as to reduce their “pathological effect,” while retaining sufficient infectious capabilities of the live virus to ensure an adaptive immune response. *Id.* As a result, the wild virus components of Flumist are “not as virulent,” giving the immune system “a little bit of an upper hand” in responding to the vaccine. *Id.* at 228.

The strength of Flumist’s immunogenicity (both in terms of localized and systemic response) has been directly studied. Tr. at 228–29; M. Barria et al., *Localized Mucosal Response to Intranasal Live Attenuated Influenza Vaccine in Adults*, J. Infectious Diseases 207:115–24 (2013), filed as Ex. A-7 (ECF No. 35-8) (“Barria”). Barria’s authors evaluated the response to Flumist in 79 subjects (aged 18–49) by evaluating blood samples taken before administration of the vaccine, and then three versus thirty days post-vaccination. Barria at 116. Barria found that Flumist provided a greater local immune response in the mucosal area of its administration, but without a corresponding “robust” systemic response, as measured by “the formation of systemic antibodies in the form of IgG.” Tr. at 229; Barria at 115.

More relevant to this case, Barria observed no change in serum cytokine profiles (in comparison to the impact of a wild viral infection) or concentrations between the date of the vaccine’s administration and three days later. Barria at 120–21; Tr. at 229–30. IL-1 $\beta$ , the cytokine most identified herein by Petitioners’ experts as causal, barely varied in amounts from before to after vaccination—and was in fact the least prevalent of the *eleven* specifically-measured cytokines. Barria at 118 (Figure 1 C). Thus, Barria’s authors concluded—contrary to a central contention of Petitioners’ experts—that the Flumist LAIV did not produce a notable systemic immune response (evidenced by increased cytokines or antibodies). Tr. at 230; Barria at 120. This kind of finding was, Dr. McCusker suggested, a basis for questioning the overall efficacy of Flumist (when coupled with other evidence establishing that the localized immune response that a LAIV like Flumist did provide was *itself* not all that effective). Tr. at 230–31.

Dr. McCusker contested Flumist’s capacity to encourage production of some of the cytokines specifically referenced by Petitioners’ experts. The causation theory offered in this case

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<sup>25</sup> Dr. Levin had deemed significant the fact that Flumist was administered *near* the brain (Tr. at 151, 152), but Dr. McCusker observed in response that this could not be so important a factor—for if it were true, then for virtually any common upper respiratory infection, “we’d be in deep trouble with every inhalation” merely due to the proximity of infection to the brain. Tr. at 291.

proposes that upregulation of IL-1 $\beta$  can promote seizures, and assumes that Flumist would trigger this upregulation. But Barria, Dr. McCusker pointed out, not only shows no unusual elevation of this cytokine overall within three days of vaccination, but it was not one of the cytokines that *was* elevated in the first place. Tr. at 233; Barria at 118. In fact, IL-1 $\beta$  would not be expected to rise in amount even in the aftermath of a wild flu virus infection, since it is not part of the usual response to such an infection. Tr. at 233. Flumist would thus not, in her view, likely encourage production of the particular proinflammatory cytokines referenced by Petitioners' experts. *Id.* at 277–78.

Overall, Dr. McCusker flatly denied the contention that Flumist was associated with seizure. Tr. at 240–41. She contrasted it with other vaccines, which she represented do report seizure as an adverse event where accompanied by fever. *Id.* at 241, 285. She saw no evidence in this case that I.R.M.'s death had any immunologic trigger. *Id.* at 283. And she questioned whether the particular cytokines most associated with administration of Flumist could cause seizure. *Id.* at 286.

Dr. McCusker next discussed what prior exposure to a particular antigen in a vaccine like Flumist would mean, immunologically, for a second, subsequent exposure. As she explained, the usual expected recall response<sup>26</sup> that a vaccine is designed to elicit in subsequent exposure to a specific infection is blunted with the flu vaccine, since its formula is regularly altered each year (in anticipation of what the coming flu season will look like). Tr. at 231. This is especially true with young children, who would likely receive a flu vaccine every year. *Id.* The record evidence in this case demonstrated that I.R.M. in fact had received the vaccine three or four times before, although Dr. McCusker could not say if the formulations did in fact differ. *Id.* at 232.

Dr. McCusker devoted some of her testimony to critiquing Petitioners' literature offered in this case. Togashi, for example, involved an acute encephalopathy precipitated by a wild infection, an experience not comparable to the impact of an attenuated virus in a vaccine (and also involving an injury not alleged in this case). Tr. at 264–66. The studied patients in Togashi, moreover, experienced fever and other acute symptoms before their CNS symptoms (which included seizure), whereas there is no evidence here that I.R.M. experienced a fever. *Id.* at 265.

Another article referenced by Petitioners as supporting their contention that LAIV components result in upregulation of proinflammatory cytokines did not discuss the specific kinds of cytokines alleged by Petitioners' experts to lower seizure thresholds—in particular IL-1 $\beta$ . Tr. at 266–69; W. Fischer et al., *Live Attenuated Influenza Vaccine Strains Elicits a Greater Innate Immune Response Than Antigenically-Matched Seasonal Influenza Viruses During Infection of*

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<sup>26</sup> Challenge-rechallenge has been described as occurring “when a person (1) is exposed to one antigen, (2) reacts to that antigen in a particular way, (3) is given the same antigen again, and (4) reacts to that antigen similarly. Typically, the second reaction is faster and more severe.” *Nussman v. Sec’y of Health & Human Servs.*, 83 Fed. Cl. 111, 119 (Fed. Cl. 2008) (internal citations omitted) (quoting *Nussman v. Sec’y of Health & Human Servs.*, No. 99-500V, 2008 WL 449656, at \*9 (Fed. Cl. Spec. Mstr. Jan. 31, 2008)); *see also Capizzano v. Sec’y of Health & Human Servs.*, 440 F.3d 1317, at 1322 (Fed. Cir. 2006) (“A rechallenge event occurs when a patient who had an adverse reaction to a vaccine suffers worsened symptoms after an additional injection of the vaccine.”).

*Human Nasal Epithelial Cell Cultures*, 32 Vaccine 15:1761–67 (2014), filed as Ex. 77 (ECF No. 71-1) (“Fischer”). Fischer did not observe an increase in IL-1 $\beta$ , and recognized that the LAIV vaccine was effective in eliciting certain chemokines *not* associated with the immune system reaction to a wild flu virus infection. Fischer at 7 (“[LAIV] vaccination likely results in a substantially different immune cell recruitment than wild type infection”). Thus, the argument that a vaccine is equivalent in effect to a milder form of wild viral infection, as Dr. Kinsbourne had suggested, was conclusory when applied to a LAIV like Flumist.

In addition, Dr. McCusker addressed the broader issue of SUDC—a topic similar to SIDS, about which she has testified on several prior occasions in Program cases. She differentiated SUDC from SIDS not only on the basis of the age classifications employed by both child’s but also “mechanistically,” noting that brain development would not be the same for a very young versus somewhat older child. Tr. at 271. In addition, recent literature on SUDC of which Dr. McCusker was aware seemed to suggest that underlying causes for the death (whether cardiac in nature, or attributable to seizure) were more easily identified. *Id.* at 271–72. The “triple-risk” model relevant to SIDS did not bear on SUDC. *Id.* at 272.

Based on her review of the literature filed in this action, Dr. McCusker unequivocally stated that there was no support for Petitioners’ contention that vaccines were a SUDC risk factor, that SUDC was dependent on an immunological trigger, or that any kind of mild infection comparable to vaccination would be associated with SUDC for children possessing a hippocampal abnormality. Tr. at 273, 283. She acknowledged, however, that it did not appear that any association between cytokine expression and SUDC has been scientifically evaluated. *Id.* And while language from Kinney I stated that “trivial infection” might constitute a SUDC risk factor, Dr. McCusker noted that in the intervening ten years that hypothesis has not been “borne out.” *Id.* Rather, the primary identified risk factors for SUDC remained sleep and sleep position, with some suggestion that febrile seizure might also explain death, based on the proposition that prior “unwitnessed seizures” that had not been diagnostically evaluated or recognized had occurred. *Id.* at 274.

### **III. Procedural History**

As noted above, this case was filed nearly five years ago. After the filing of the statement of completion, I ordered Petitioners to begin filing expert reports, with Respondent’s Rule 4(c) Report to come after. Docket Entry Scheduling Order, dated October 27, 2015. Petitioners complied with the Order, filing Drs. Kinsbourne’s and Levin’s initial reports in December 2015. Respondent subsequently filed his Rule 4(c) Report in January 2016, maintaining that an entitlement award was not appropriate.

Once Petitioners located and filed some additional medical records Respondent had identified as missing, Respondent filed expert reports from Drs. McCusker and Harris in December 2016. In reaction, Petitioners filed supplemental reports from Drs. Levin and Kinsbourne in May

2017. I subsequently held a status conference with the parties, and at that time informed Petitioners that I had doubts about the persuasiveness of their theory. Order, dated July 31, 2017 (ECF No. 47). I also noted the parallels between the arguments advanced in this case and those that had been routinely rejected in SIDS claims before, although I acknowledged that the SIDS classification did not literally apply to I.R.M. *Id.* I ordered Petitioners to consider filing a supplemental expert report.

After considerable delay, Petitioners filed Dr. Miller's expert report in April 2018. A little before, however, I set this matter down for an entitlement hearing to be held in May 2019. Prehearing Order, dated February 20, 2018 (ECF No. 59). Before the hearing, the parties submitted briefs and some additional evidence (including a supplemental report from Dr. Harris responding to Dr. Miller), and the hearing proceeded as scheduled.

In September 2019, both sides filed post-hearing briefs. Respondent also filed a supplemental report from Dr. McCusker (*see generally* ECF No. 79) - despite the fact that my prehearing order in this case set a deadline of April 11, 2019 to file such materials. Order, dated February 20, 2018 (ECF No. 59).<sup>27</sup> This four-page report sets forth Dr. McCusker's reaction to certain articles and arguments pertaining to testimony mostly provided by Dr. Miller about cytokines and how they might arguably travel into the brain and thereby cause injury. Objecting to the untimeliness of this filing, Petitioners requested that it be stricken. Motion to Strike, dated September 30, 2019 (ECF No. 81). Respondent, in turn, argued that the supplemental report was intended to address six additional items of literature filed post-trial by Petitioners (*see* ECF No. 76) (articles filed July 9, 2019), and thus fairness dictated that Respondent either be permitted to react to the items or that they be stricken as well. Opposition, dated October 7, 2019 (ECF No. 83). (My disposition of the Motion to Strike is set forth below).

The matter is now ripe for resolution.

#### **IV. Applicable Legal Standards**

##### *A. Petitioner's Overall Burden in Vaccine Program Cases*

To receive compensation in the Vaccine Program, a petitioner must prove either: (1) that he suffered a "Table Injury"—i.e., an injury falling within the Vaccine Injury Table—corresponding to one of the vaccinations in question within a statutorily prescribed period of time or, in the alternative, (2) that his illnesses were actually caused by a vaccine (a "Non-Table Injury"). *See* Sections 13(a)(1)(A), 11(c)(1), and 14(a), as amended by 42 C.F.R. § 100.3; § 11(c)(1)(C)(ii)(I); *see also Moberly v. Sec'y of Health & Human Servs.*, 592 F.3d 1315, 1321 (Fed.

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<sup>27</sup> Dr. Harris's supplemental report, though filed not long before the hearing, was nevertheless in compliance with this deadline.

Cir. 2010); *Capizzano v. Sec’y of Health & Human Servs.*, 440 F.3d 1317, 1320 (Fed. Cir. 2006).<sup>28</sup> In this case, Petitioner does not assert a Table claim.

For both Table and Non-Table claims, Vaccine Program petitioners bear a “preponderance of the evidence” burden of proof. Section 13(1)(a). That is, a petitioner must offer evidence that leads the “trier of fact to believe that the existence of a fact is more probable than its nonexistence before [he] may find in favor of the party who has the burden to persuade the judge of the fact’s existence.” *Moberly*, 592 F.3d at 1322 n.2; *see also Snowbank Enter. v. United States*, 6 Cl. Ct. 476, 486 (1984) (mere conjecture or speculation is insufficient under a preponderance standard). Proof of medical certainty is not required. *Bunting v. Sec’y of Health & Human Servs.*, 931 F.2d 867, 873 (Fed. Cir. 1991). In particular, a petitioner must demonstrate that the vaccine was “not only [the] but-for cause of the injury but also a substantial factor in bringing about the injury.” *Moberly*, 592 F.3d at 1321 (quoting *Shyface v. Sec’y of Health & Human Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)); *Pafford v. Sec’y of Health & Human Servs.*, 451 F.3d 1352, 1355 (Fed. Cir. 2006). A petitioner may not receive a Vaccine Program award based solely on his assertions; rather, the petition must be supported by either medical records or by the opinion of a competent physician. Section 13(a)(1).

In attempting to establish entitlement to a Vaccine Program award of compensation for a Non-Table claim, a petitioner must satisfy all three of the elements established by the Federal Circuit in *Althen*: “(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of proximate temporal relationship between vaccination and injury.” *Althen*, 418 F.3d at 1278.

Each of the *Althen* prongs requires a different showing. Under *Althen* prong one, petitioners must provide a “reputable medical theory,” demonstrating that the vaccine received *can cause* the type of injury alleged. *Pafford*, 451 F.3d at 1355–56 (citations omitted). To satisfy this prong, a petitioner’s theory must be based on a “sound and reliable medical or scientific explanation.” *Knudsen v. Sec’y of Health & Human Servs.*, 35 F.3d 543, 548 (Fed. Cir. 1994). Such a theory must only be “legally probable, not medically or scientifically certain.” *Id.* at 549.

Petitioners may satisfy the first *Althen* prong without resort to medical literature, epidemiological studies, demonstration of a specific mechanism, or a generally accepted medical theory. *Andreu v. Sec’y of Health & Human Servs.*, 569 F.3d 1367, 1378–79 (Fed. Cir. 2009) (citing *Capizzano*, 440 F.3d at 1325–26). Special masters, despite their expertise, are not

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<sup>28</sup> Decisions of special masters (some of which I reference in this ruling) constitute persuasive but not binding authority. *Hanlon v. Sec’y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec’y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff’d* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec’y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at \*7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014).

empowered by statute to conclusively resolve what are essentially thorny scientific and medical questions, and thus scientific evidence offered to establish *Althen* prong one is viewed “not through the lens of the laboratorian, but instead from the vantage point of the Vaccine Act’s preponderant evidence standard.” *Id.* at 1380. Accordingly, special masters must take care not to increase the burden placed on petitioners in offering a scientific theory linking vaccine to injury. *Contreras*, 121 Fed. Cl. at 245 (“[p]lausibility . . . in many cases *may* be enough to satisfy *Althen* prong one” (emphasis in original)).

In discussing the evidentiary standard applicable to the first *Althen* prong, the Federal Circuit has consistently rejected the contention that it can be satisfied merely by establishing a proposed causal theory’s scientific or medical *plausibility*. See *Boatmon v. Sec’y of Health & Human Servs.*, 941 F.3d 1351, 1359 (Fed. Cir. 2019); see also *LaLonde v. Sec’y of Health & Human Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (“[h]owever, in the past we have made clear that simply identifying a ‘plausible’ theory of causation is insufficient for a petitioner to meet her burden of proof.” (citing *Moberly*, 592 F.3d at 1322)). Thus, the same preponderance standard used overall in evaluating a claimant’s success in a Vaccine Act claim is also applied specifically to the first *Althen* prong. See, e.g., *Broekelschen*, 618 F.3d at 1350 (affirming special master’s determination that expert “had not provided a ‘reliable medical or scientific explanation’ *sufficient to prove by a preponderance of the evidence a medical theory* linking the [relevant vaccine to relevant injury].”) (emphasis added). And petitioners always have the ultimate burden of establishing their Vaccine Act claim *overall* with preponderant evidence. *W.C. v. Sec’y of Health & Human Servs.*, 704 F.3d 1352, 1356 (Fed. Cir. 2013) (citations omitted); *Tarsell*, 133 Fed. Cl. at 793 (noting that *Moberly* “addresses the petitioner’s overall burden of proving causation-in-fact under the Vaccine Act” by a preponderance standard)<sup>29</sup>.

The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner’s medical records. *Althen*, 418 F.3d at 1278; *Andreu*, 569 F.3d at 1375–77; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec’y of Health & Human Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). In establishing that a vaccine “did cause” injury, the opinions and views of the injured party’s treating physicians are entitled to some weight. *Andreu*, 569 F.3d at 1367; *Capizzano*, 440 F.3d at 1326 (“medical records and medical opinion testimony are favored in vaccine cases, as treating physicians are likely to be in the best position to determine whether a ‘logical sequence of cause and effect show[s] that the vaccination was the reason for the injury’”) (quoting *Althen*, 418 F.3d at 1280). Medical records are generally viewed as particularly trustworthy evidence, since they are created contemporaneously with the treatment of the patient. *Cucuras v. Sec’y of Health & Human Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993).

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<sup>29</sup> Although decisions like *Contreras* suggest that the burden of proof required to satisfy the first *Althen* prong is less stringent than the other two, there is ample contrary authority for the more straightforward proposition that when considering the first prong, the same preponderance standard used overall is also applied when evaluating if a reliable and plausible causal theory has been established. *Broekelschen*, 618 F.3d at 1350.

Medical records and statements of a treating physician, however, do not *per se* bind the special master to adopt the conclusions of such an individual, even if they must be considered and carefully evaluated. Section 13(b)(1) (providing that “[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court”); *Snyder v. Sec’y of Health & Human Servs.*, 88 Fed. Cl. 706, 746 n.67 (2009) (“there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted”). As with expert testimony offered to establish a theory of causation, the opinions or diagnoses of treating physicians are only as trustworthy as the reasonableness of their suppositions or bases. The views of treating physicians should be weighed against other, contrary evidence also present in the record—including conflicting opinions among such individuals. *Hibbard v. Sec’y of Health & Human Servs.*, 100 Fed. Cl. 742, 749 (2011) (not arbitrary or capricious for special master to weigh competing treating physicians’ conclusions against each other), *aff’d*, 698 F.3d 1355 (Fed. Cir. 2012); *Veryzer v. Sec’y of Dept. of Health & Human Servs.*, No. 06-522V, 2011 WL 1935813, at \*17 (Fed. Cl. Spec. Mstr. Apr. 29, 2011), *mot. for review denied*, 100 Fed. Cl. 344, 356 (2011), *aff’d without opinion*, 475 F. Appx. 765 (Fed. Cir. 2012).

The third *Althen* prong requires establishing a “proximate temporal relationship” between the vaccination and the injury alleged. *Althen*, 418 F.3d at 1281. That term has been equated to the phrase “medically-acceptable temporal relationship.” *Id.* A petitioner must offer “preponderant proof that the onset of symptoms occurred within a timeframe which, given the medical understanding of the disorder’s etiology, it is medically acceptable to infer causation.” *de Bazan v. Sec’y of Health & Human Servs.*, 539 F.3d 1347, 1352 (Fed. Cir. 2008). The explanation for what is a medically acceptable timeframe must align with the theory of how the relevant vaccine can cause an injury (*Althen* prong one’s requirement). *Id.* at 1352; *Shapiro v. Sec’y of Health & Human Servs.*, 101 Fed. Cl. 532, 542 (2011), *recons. denied after remand*, 105 Fed. Cl. 353 (2012), *aff’d mem.*, 503 F. App’x. 952 (Fed. Cir. 2013); *Koehn v. Sec’y of Health & Human Servs.*, No. 11-355V, 2013 WL 3214877 (Fed. Cl. Spec. Mstr. May 30, 2013), *mot. for rev. denied* (Fed. Cl. Dec. 3, 2013), *aff’d*, 773 F.3d 1239 (Fed. Cir. 2014).

#### B. Legal Governing Factual Determinations

The process for making determinations in Vaccine Program cases regarding factual issues begins with consideration of the medical records. Section 11(c)(2). The special master is required to consider “all [] relevant medical and scientific evidence contained in the record,” including “any diagnosis, conclusion, medical judgment, or autopsy or coroner’s report which is contained in the record regarding the nature, causation, and aggravation of the petitioner’s illness, disability, injury, condition, or death,” as well as the “results of any diagnostic or evaluative test which are contained in the record and the summaries and conclusions.” Section 13(b)(1)(A). The special master is then required to weigh the evidence presented, including contemporaneous medical records and testimony. *See Burns v. Sec’y of Health & Human Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993) (it is

within the special master's discretion to determine whether to afford greater weight to contemporaneous medical records than to other evidence, such as oral testimony surrounding the events in question that was given at a later date, provided that such determination is evidenced by a rational determination).

Medical records that are created contemporaneously with the events they describe are presumed to be accurate and "complete" (i.e., presenting all relevant information on a patient's health problems). *Cucuras*, 993 F.2d at 1528; *Doe/70 v. Sec'y of Health & Human Servs.*, 95 Fed. Cl. 598, 608 (2010) ("[g]iven the inconsistencies between petitioner's testimony and his contemporaneous medical records, the special master's decision to rely on petitioner's medical records was rational and consistent with applicable law"), *aff'd sub nom. Rickett v. Sec'y of Health & Human Servs.*, 468 F. Appx. 952 (Fed. Cir. 2011) (non-precedential opinion). This presumption is based on the linked propositions that (i) sick people visit medical professionals; (ii) sick people honestly report their health problems to those professionals; and (iii) medical professionals record what they are told or observe when examining their patients in as accurate a manner as possible, so that they are aware of enough relevant facts to make appropriate treatment decisions. *Sanchez v. Sec'y of Health & Human Servs.*, No. 11-685V, 2013 WL 1880825, at \*2 (Fed. Cl. Spec. Mstr. Apr. 10, 2013), *rev'd on other grounds*, 2020 WL 1685554, --- F. App'x --- (Fed. Cir. 2020); *Cucuras v. Sec'y of Health & Human Servs.*, 26 Cl. Ct. 537, 543 (1992), *aff'd*, 993 F.2d at 1525 (Fed. Cir. 1993) ("[i]t strains reason to conclude that petitioners would fail to accurately report the onset of their daughter's symptoms").

Accordingly, if the medical records are clear, consistent, and complete, then they should be afforded substantial weight. *Lowrie*, 2005 WL 6117475, at \*20. Indeed, contemporaneous medical records are generally found to be deserving of greater evidentiary weight than oral testimony—especially where such testimony conflicts with the record evidence. *Cucuras*, 993 F.2d at 1528; *see also Murphy*, 23 Cl. Ct. at 733 (citing *United States v. United States Gypsum Co.*, 333 U.S. 364, 396 (1947) ("[i]t has generally been held that oral testimony which is in conflict with contemporaneous documents is entitled to little evidentiary weight.")).

There are, however, situations in which compelling oral testimony may be more persuasive than written records, such as where records are deemed to be incomplete or inaccurate. *Campbell v. Sec'y of Health & Human Servs.*, 69 Fed. Cl. 775, 779 (2006) ("like any norm based upon common sense and experience, this rule should not be treated as an absolute and must yield where the factual predicates for its application are weak or lacking"); *Lowrie*, 2005 WL 6117475, at \*19 ("[w]ritten records which are, themselves, inconsistent, should be accorded less deference than those which are internally consistent") (quoting *Murphy*, 23 Cl. Ct. at 733)). Ultimately, a determination regarding a witness's credibility is needed when determining the weight that such testimony should be afforded. *Andreu*, 569 F.3d at 1379; *Bradley v. Sec'y of Health & Human Servs.*, 991 F.2d 1570, 1575 (Fed. Cir. 1993).

When witness testimony is offered to overcome the presumption of accuracy afforded to contemporaneous medical records, such testimony must be “consistent, clear, cogent, and compelling.” *Sanchez*, 2013 WL 1880825, at \*3 (citing *Blutstein v. Sec’y of Health & Human Servs.*, No. 90-2808V, 1998 WL 408611, at \*5 (Fed. Cl. Spec. Mstr. June 30, 1998)). In determining the accuracy and completeness of medical records, the Court of Federal Claims has listed four possible explanations for inconsistencies between contemporaneously created medical records and later testimony: (1) a person’s failure to recount to the medical professional everything that happened during the relevant time period; (2) the medical professional’s failure to document everything reported to her or him; (3) a person’s faulty recollection of the events when presenting testimony; or (4) a person’s purposeful recounting of symptoms that did not exist. *Lalonde v. Sec’y of Health & Human Servs.*, 110 Fed. Cl. 184, 203-04 (2013), *aff’d*, 746 F.3d 1334 (Fed. Cir. 2014). In making a determination regarding whether to afford greater weight to contemporaneous medical records or other evidence, such as testimony at hearing, there must be evidence that this decision was the result of a rational determination. *Burns*, 3 F.3d at 417.

### C. Analysis of Expert Testimony

Establishing a sound and reliable medical theory often requires a petitioner to present expert testimony in support of his claim. *Lampe v. Sec’y of Health & Human Servs.*, 219 F.3d 1357, 1361 (Fed. Cir. 2000). Vaccine Program expert testimony is usually evaluated according to the factors for analyzing scientific reliability set forth in *Daubert v. Merrell Dow Pharmaceuticals, Inc.*, 509 U.S. 579, 594–96 (1993). See *Cedillo v. Sec’y of Health & Human Servs.*, 617 F.3d 1328, 1339 (Fed. Cir. 2010) (citing *Terran v. Sec’y of Health & Human Servs.*, 195 F.3d 1302, 1316 (Fed. Cir. 1999)). “The *Daubert* factors for analyzing the reliability of testimony are: (1) whether a theory or technique can be (and has been) tested; (2) whether the theory or technique has been subjected to peer review and publication; (3) whether there is a known or potential rate of error and whether there are standards for controlling the error; and (4) whether the theory or technique enjoys general acceptance within a relevant scientific community.” *Terran*, 195 F.3d at 1316 n.2 (citing *Daubert*, 509 U.S. at 592–95).

The *Daubert* factors play a slightly different role in Vaccine Program cases than they do when applied in other federal judicial fora (such as the district courts). *Daubert* factors are usually employed by judges (in the performance of their evidentiary gatekeeper roles) to exclude evidence that is unreliable and/or could confuse a jury. In Vaccine Program cases, by contrast, these factors are used in the *weighing* of the reliability of scientific evidence proffered. *Davis v. Sec’y of Health & Human Servs.*, 94 Fed. Cl. 53, 66–67 (2010) (“uniquely in this Circuit, the *Daubert* factors have been employed also as an acceptable evidentiary-gauging tool with respect to persuasiveness of expert testimony already admitted”). The flexible use of the *Daubert* factors to evaluate the persuasiveness and reliability of expert testimony has routinely been upheld. See, e.g., *Snyder*, 88

Fed. Cl. at 742–45. In this matter (as in numerous other Vaccine Program cases), *Daubert* has not been employed at the threshold, to determine what evidence should be admitted, but instead to determine whether expert testimony offered is reliable and/or persuasive.

Respondent frequently offers one or more experts of his own in order to rebut a petitioner's case. Where both sides offer expert testimony, a special master's decision may be “based on the credibility of the experts and the relative persuasiveness of their competing theories.” *Broekelschen*, 618 F.3d at 1347 (citing *Lampe*, 219 F.3d at 1362). However, nothing requires the acceptance of an expert's conclusion “connected to existing data only by the *ipse dixit* of the expert,” especially if “there is simply too great an analytical gap between the data and the opinion proffered.” *Snyder*, 88 Fed. Cl. at 743 (quoting *Gen. Elec. Co. v. Joiner*, 522 U.S. 136, 146 (1997)); see also *Isaac v. Sec'y of Health & Human Servs.*, No. 08-601V, 2012 WL 3609993, at \*17 (Fed. Cl. Spec. Mstr. July 30, 2012), *mot. for rev. denied*, 108 Fed. Cl. 743 (2013), *aff'd*, 540 F. Appx. 999 (Fed. Cir. 2013) (citing *Cedillo*, 617 F.3d at 1339). Weighing the relative persuasiveness of competing expert testimony, based on a particular expert's credibility, is part of the overall reliability analysis to which special masters must subject expert testimony in Vaccine Program cases. *Moberly*, 592 F.3d at 1325–26 (“[a]ssessments as to the reliability of expert testimony often turn on credibility determinations”); see also *Porter v. Sec'y of Health & Human Servs.*, 663 F.3d 1242, 1250 (Fed. Cir. 2011) (“this court has unambiguously explained that special masters are expected to consider the credibility of expert witnesses in evaluating petitions for compensation under the Vaccine Act”).

Expert opinions based on unsupported facts may be given relatively little weight. See *Dobrydnev v. Sec'y of Health & Human Servs.*, 556 F. Appx. 976, 992–93 (Fed. Cir. 2014) (“[a] doctor's conclusion is only as good as the facts upon which it is based”) (citing *Brooke Group Ltd. v. Brown & Williamson Tobacco Corp.*, 509 U.S. 209, 242 (1993) (“[w]hen an expert assumes facts that are not supported by a preponderance of the evidence, a finder of fact may properly reject the expert's opinion”). Expert opinions that fail to address or are at odds with contemporaneous medical records may therefore be less persuasive than those which correspond to such records. See *Gerami v. Sec'y of Health & Human Servs.*, No. 12-442V, 2013 WL 5998109, at \*4 (Fed. Cl. Spec. Mstr. Oct. 11, 2013), *mot. for review den'd*, 127 Fed. Cl. 299 (2014).

#### D. *Consideration of Medical Literature*

Both parties filed medical and scientific literature in this case, but not every filed item factors into the outcome of this decision. While I have reviewed all the medical literature submitted in this case, I discuss only those articles that are most relevant to my determination and/or are central to Petitioner's case—just as I have not exhaustively discussed every individual medical record filed. *Moriarty v. Sec'y of Health & Human Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) (“[w]e generally presume that a special master considered the relevant record evidence even

though he does not explicitly reference such evidence in his decision”) (citation omitted); *see also Paterek v. Sec’y of Health & Human Servs.*, 527 F. Appx. 875, 884 (Fed. Cir. 2013) (“[f]inding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered”).

#### E. *Consideration of Comparable Special Master Decisions*

In reaching a decision in this case, I have taken into account other decisions issued by special masters involving similar injuries, vaccines, or circumstances. I also reference some of those cases in this Decision, in an effort to establish common themes, as well as demonstrate how prior determinations impact my thinking on the present case.

There is no error in doing so. It is certainly correct that prior decision in different cases do not *control* the outcome herein.<sup>30</sup> *Boatmon*, 941 F.3d at 1358-59; *Hanlon v. Sec’y of Health & Human Servs.*, 40 Fed. Cl. 625, 630 (1998). Thus, the fact that another special master reasonably determined elsewhere, on the basis of facts not in evidence in this case, that preponderant evidence supported the conclusion that vaccine X caused petitioner’s injury Y does not compel me to reach the same conclusion in *this* case. Different actions present different background medical histories, different experts, and different items of medical literature, and therefore can reasonably result in contrary determinations.

However, it is *equally* the case that special masters reasonably draw upon their experience in resolving Vaccine Act claims. *Doe v. Sec’y of Health & Human Servs.*, 76 Fed. Cl. 328, 338–39 (2007) (“[o]ne reason that proceedings are more expeditious in the hands of special masters is that the special masters have the *expertise and experience to know the type of information that is most probative of a claim*”) (emphasis added). They would be remiss in ignoring prior cases presenting similar theories or factual circumstances, along with the reasoning employed in reaching such decisions. This is especially so given that special masters not only routinely hear from the same experts in comparable cases, but are also repeatedly offered the *same* items of medical literature regarding certain common causation theories. It defies reason and logic to obligate special masters to “reinvent the wheel”, so to speak, in each new case before them, paying no heed at all to how their colleagues past and present have addressed similar causation theories or fact patterns.

It is for this reason that prior decisions can have high persuasive value—and why special

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<sup>30</sup> By contrast, Federal Circuit rulings concerning legal issues are binding on special masters. *Guillory v. Sec’y of Health & Human Servs.*, 59 Fed. Cl. 121, 124 (2003), *aff’d* 104 F. Appx. 712 (Fed. Cir. 2004); *see also Spooner v. Sec’y of Health & Human Servs.*, No. 13-159V, 2014 WL 504728, at \*7 n.12 (Fed. Cl. Spec. Mstr. Jan. 16, 2014). Special masters are also bound within a specific case by determinations made by judges of the Court of Federal Claims after a motion for review is resolved.

masters often explain how a new determination relates to such past decisions.<sup>31</sup> Even if the Federal Circuit does not *require* special masters to distinguish other relevant cases (*Boatmon*, 941 F.3d at 1358), it is still *wise* to do so.

## ANALYSIS

### **I. Petitioners' Causation Theories are Unreliable and Unpersuasive<sup>32</sup>**

#### *A. Prior Relevant Decisions – SIDS and Vaccine-Induced Seizure*

Special masters have had numerous opportunities to evaluate whether vaccines can cause unexplained death via SIDS in infants younger than I.R.M. They have almost never found so, and their determinations have been consistently upheld on appeal. *See, e.g., Cozart v. Sec'y of Health & Human Servs.*, No. 00–590V, 2015 WL 6746616, at \*1 (Fed. Cl. Spec. Mstr. Oct. 15, 2015), *mot. for review den'd*, 126 Fed. Cl. 488 (2016); *Nunez v. Sec'y of Health & Human Servs.*, No. 14–863V, 2019 WL 2462667, at \*1 (Fed. Cl. Spec. Mstr. Mar. 29, 2019), *mot. for rev. den'd*, 144 Fed. Cl. 540 (Fed. Cl. 2019), *appeal docketed*, No. 20-1021 (Fed. Cir. Oct. 8, 2019). Those prior cases also uniformly featured Drs. Miller and McCusker—underscoring the parallel nature of the present claim. *See, e.g., Cozart*, 2015 WL 6746616, at \*9–11. Their views have been evaluated and demeanors considered time and time again—but Respondent has always prevailed.

In only one instance has a special master found for a petitioner in a SIDS case—but that determination was reversed by the Court of Federal Claims, with the Federal Circuit affirming the reversal. *Boatmon*, 941 F.3d at 1353. In so ruling, the Federal Circuit found that Dr. Miller's theories about the purported role cytokines could play in causing or contributing to a SIDS death (theories paralleling the arguments offered in this case) were not supported with sufficient reliable science, relying less on establishing how cytokines would function as opposed to the fact that cytokines were *present* in association with certain brain injuries, or could cross the blood-brain

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<sup>31</sup> Consideration of prior determinations is a two-way street that does not only inure to the benefit of one party. Thus, I would likely take into account the numerous decisions finding no association between vaccination and autism when confronted with a new claim asserting autism as an injury, and have informed such claimants early in the life of their case that the claim was not viable for just that reason. But I would *also* deem a non-Table claim asserting GBS after the flu vaccine as not requiring extensive proof on *Althen* prong one “can cause” matters, for the simple reason that the Program has repeatedly litigated the issue in favor of petitioners.

<sup>32</sup> My opinion almost wholly turns on the first two *Althen* prongs, so I do not also include an extended discussion of Petitioners' success at demonstrating that the timeframe for alleged onset in this case was medically acceptable. *K.L. v. Sec'y of Health & Human Servs.*, No. 12-312V, 2017 WL 1713110, at \*16 (Fed. Cl. Spec. Mstr. Mar. 17, 2017), *mot. for rev. denied*, 134 Fed. Cl. 579 (Fed. Cl. 2018). I do note, however, that although the timeframe from vaccination to I.R.M.'s tragic death was consistent with Petitioners' theory (and particularly was within the two-to-three day time period in which *some* proinflammatory cytokines produced in response to Flumist might upregulate via an innate immune response), Petitioners did *not* preponderantly demonstrate (a) that this actually occurred in I.R.M.'s case after the administration of Flumist, or (b) that the one cytokine most referenced by Petitioners' experts, IL-1 $\beta$ , is upregulated after Flumist administration at all, let alone in sufficient quantities to be pathologic.

barrier under specific circumstances. *Id.* at 1360–62. The *Boatmon* claimants also could not substantiate the more fundamental contention—that vaccines could play *any* role in the SIDS “triple risk” model. *Id.* at 1360.

I acknowledge the distinctions between the causation theories offered herein, which relate to an instance of SUDC, from those mounted in SIDS cases. All of the experts testifying in this case agreed that SIDS and SUDC are not congruent, and that the “triple risk” model employed in SIDS circumstances has no direct application here, since it is believed by medical science that the incomplete development of the brains of very young infants plays a role in their susceptibility to the SIDS risk factors. Moreover, one specific element of the SIDS theory that has been repeatedly rejected—that immune system interaction with an infant’s brain stem causes respiratory failure associated with SIDS, thereby rendering vaccination a risk factor itself—is not at issue in this case at all. *Compare Boatmon*, 138 Fed. Cl. at 569.

**I thus do not conclude that the many SIDS cases going against petitioners in the Vaccine Program compelled the same result herein.** The fact that I held a hearing in this case, *despite* my concerns about the similarity of this case to the prior SIDS determinations, and have written a lengthy decision evaluating the arguments asserted, should underscore the degree to which I have tried to give Petitioners’ claim a fair shot at success.

Nevertheless - this claim, like the prior SIDS cases, relies on the theory that vaccine-induced cytokine interference with the brain in some way has pathologic, and ultimately fatal, outcomes under circumstances involving very young children that otherwise remain mysterious to medical science. And in such comparable cases, special masters have repeatedly noted that existing medical and scientific evidence does not reliably support the contention that cytokines *cause* such processes - as opposed to appear in *response* to an ongoing pathogenic process caused by something else. It was for such reasons that the Federal Circuit in *Boatmon* termed the causation theory therein offered as merely “plausible”—and therefore insufficient to meet the preponderant test. *Boatmon*, 941 F.3d at 1360.<sup>33</sup> My decision below reasonably takes such parallel analyses into account.

There are also several decisions in which special masters have found that a vaccine could induce an initial seizure in an infant or very young child. Most commonly, they have reached such conclusions after the child experienced a fever in response to the vaccine. *See, e.g., Graves v. Sec’y of Health & Human Servs.*, 109 Fed. Cl. 579 (Fed. Cl. 2013) (summarizing that petitioners were entitled to compensation when a Prevnar vaccine caused their daughter to suffer from seizures after experiencing a fever). In effect, these findings stand for the proposition that the innate immune response to vaccination, and not a specific adaptive-autoimmune process (in which

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<sup>33</sup> I note that Respondent filed the Federal Circuit’s *Boatmon* decision as an exhibit in this case after its release, perhaps in the hope that I would treat it as dispositive herein. Notice of Additional Authority, filed on November 8, 2019 (ECF No. 84). The preceding discussion, however, should make clear that this is not so—this is not *literally* a “SIDS case.”

specific components of the vaccine interact with self structures, or otherwise induce seizure)<sup>34</sup>, can produce a fever which triggers an initial seizure, thereby propelling the child into a chain of ever-more-damaging seizures thereafter. *Fuller v. Sec’y of Health & Human Servs.*, No. 15-1470V, 2019 WL 7576382, at \*18 (Fed. Cl. Spec. Mstr. Dec. 17, 2019). Decisions going the other way have also been issued, but typically where there is some persuasive alternative explanation for the seizure, such as a preexisting genetic disorder. *Oliver v. Sec’y of Health & Human Servs.*, No. 10-394V, 2017 WL 747846, at \*2 (Fed. Cl. Spec. Mstr. Feb. 1, 2017) (determining that SCN1A gene mutation, and not vaccination, explained child’s Dravet syndrome), *mot. for review den’d*, 133 Fed. Cl. 341, 344 (Fed. Cl. 2017), *aff’d*, 900 F.3d 1357, 1359 (Fed. Cir. 2018).

**B. *Petitioners Have Not Preponderantly Established that the Flumist Vaccine Can Trigger Non-Febrile Seizures in a Susceptible Child Through an Innate Systemic Inflammatory Process***

Petitioners’ experts shifted their causation theory somewhat over the course of the proceedings.<sup>35</sup> Although Drs. Kinsbourne and Levin initially pointed to brain edema triggered by vaccination as causal, at hearing they maintained that (1) a child with a hippocampal abnormality like I.R.M. was susceptible to seizure, (2) the conditions of sleep made seizure more likely under such circumstances, (3) a seizure experienced while sleeping could result in unexplained death, and (4) the Flumist vaccine was in particular likely to further increase the seizure risk due to its stimulation of the innate immune system. While items (1) to (3) were persuasively established with reliable science (and largely not contested by Respondent’s experts), the fourth element of the theory, and the component most critical to Petitioners’ success, was not.

Both sides’ primary pathologists (Drs. Miller and Harris) were credentialed and credible, and they agreed that a hippocampal malformation was likely associated with childhood seizures—especially during sleep. They also concurred that literature on SUDC ties these factors together (although, as Dr. Harris noted, in many SUDC cases a seizure-induced fatality was, or appeared to have been, preceded by seizures that went unrecognized—and therefore such cases would closely align with SUDEP, but for the absence of a prior epilepsy diagnosis). Tr. at 17–18, 186. But there

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<sup>34</sup> Some decisions have suggested that the components of a particular vaccine could cause a seizure independently of fever. See *Graves v. Sec’y of Health & Human Servs.*, 101 Fed. Cl. 310, 339 (Fed. Cl. 2011) (Prevnam vaccine). However, other decisions have noted that changes in vaccine formulation have greatly reduced the likelihood of such a reaction—and in turn called into question the reasoning of such other cases. See *Sharpe v. Sec’y of Health & Human Servs.*, No. 14-65V, 2018 WL 7625360, at \*32 (Fed. Cl. Spec. Mstr. Nov. 5, 2018) (“Subsequent decisions have underscored that the DTaP vaccine’s injury-causing potential cannot be conflated with findings pertinent only to the DPT whole-cell version.”).

<sup>35</sup> Before the filing of Dr. Miller’s report, Drs. Levin and Kinsbourne largely opined that the Flumist vaccine caused I.R.M.’s brain edema, and that this was the likely cause of his death. Levin Rep. at 7–8; Kinsbourne Rep. at 3. After Dr. Miller weighed in, however, Dr. Kinsbourne revised his opinion to be consistent. Kinsbourne Supp. Rep. at 6; Tr. at 73, 99. At hearing, Dr. Levin seemed to accept this altered theory, although he continued to embrace the contention in his reports that the vaccine could also cause brain edema. Tr. at 153, 155. As discussed below, this expert contention was especially unpersuasive—although my rejection of it did not eliminate all causal theories offered in this case.

is little to no direct evidence that any vaccine, let alone the Flumist vaccine, could cause or contribute to<sup>36</sup> this process, nor could any of Petitioners' experts speak from their own experience, whether from patient treatment or research, to bulwark this point.

Petitioners instead attempted to support this aspect of their theory by connecting several indirect items of proof (of course a valid means of establishing entitlement in the Program). But important sections of this chain of opinion were medically/scientifically unreliable. The most significant insufficient element was the lack of evidence pertaining to the direct or initial pathologic capacity of cytokines. As noted above, the inability to preponderantly establish this particular causal element has resulted in the dismissal of *all* prior SIDS claims. In addition, I have on many occasions considered whether the transient upregulation of cytokines<sup>37</sup> attributable to the innate immune system's initial response to a vaccine can be pathologic, but have consistently found this contention could not be substantiated with reliable scientific or medical evidence.

For example, Kashiwagi—an article mentioned by both sides at hearing – is an item of literature I have discussed at length in other decisions. *See, e.g., Dean v. Sec'y of Health & Human Servs.*, No. 13-808V, 2017 WL 2926605, at \*17 (Fed. Cl. Spec. Mstr. June 9, 2017) (DTaP and Hib vaccines did not cause child's neurologic deficits).<sup>38</sup> Kashiwagi only observes the transient

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<sup>36</sup> Petitioners did not articulate a significant aggravation claim, nor did they describe I.R.M.'s brain malformation (which the facts do suggest predated vaccination) as a preexisting problem worsened by vaccination, so I do not include herein a significant aggravation analysis.

<sup>37</sup> Circumstances might be different if the record supported the conclusion that a "cytokine storm" had occurred, but Petitioners' experts plainly stated they did not believe this to have occurred herein. Tr. at 157 (Dr. Levin, Petitioners' sole testifying immunologist, admitting I.R.M. did not experience a cytokine storm). But even that kind of circumstance will typically occur in response to an *ongoing and uncontrolled infectious process*. *See* Geoff Brumfiel *Why Some COVID-19 Patients Crash*, NPR (April 7, 2020) <https://www.npr.org/sections/health-shots/2020/04/07/828091467/why-some-covid-19-patients-crash-the-bodys-immune-system-might-be-to-blame> (explaining that a cytokine storm is the immune system's persistent overreaction to a disease). No persuasive evidence or testimony offered in this case established that the Flumist vaccine could set into motion a cytokine storm process.

<sup>38</sup> In *Dean*, I specifically commented on Kashigawi as follows:

Not only was Kashiwagi's study not designed to examine the effects of cytokines in the brain following vaccination, but its central purpose (comparing the levels of inflammatory cytokines in the sera of vaccine recipients with febrile and non-febrile illnesses within 24 hours of vaccination) does not shed light on whether the particular kinds of cytokines produced in the study could cause the injury proposed herein (a CNS-oriented injury, as opposed to peripheral cytokine changes occurring at the locus of vaccination).

Beyond the above, neither Kashiwagi nor any other literature that Petitioners cite establishes that cytokine upregulation could be maintained biologically for long enough, and in sufficient quantities as well, to act as [petitioner's] theory proposes. In fact, Kashiwagi's study shows that cytokine production increased only for approximately 24 hours following stimulation. . . . Further, the only increased cytokine identified in the serum of the test subjects was an elevated G-CSF level in individuals with a febrile illness, and the authors were unable to determine the significance of this result. *Id.* The decisions of other special masters (albeit not in precisely the same circumstances) have noted that Kashiwagi does not support the idea that cytokines produced in response to vaccination could negatively impact the brain. *See, e.g., Copenhaver v. Sec'y of Health & Human*

upregulation of *some* proinflammatory cytokines (not including IL-1 $\beta$ )—not that they are also pathogenic. It also did not test *any* version of the flu vaccine, LAIV or otherwise. Kashiwagi thus does not stand for the conclusion that the expected and intended effect of immune stimulation caused by vaccination can turn pathologic. Togashi is similarly unpersuasive as proof for Petitioners’ causation theory. Not only did it involve an injury not alleged herein (acute encephalopathy), but it did not firmly conclude whether cytokines were initially causal, or merely contributed to an ongoing pathologic process attributable to a direct wild flu virus infection (and the article ultimately concluded that vaccination was the best means of preventing the infection—directly contrary to the proposition that vaccination herein instigated a pathologic process). Togashi at 78.

In addition, some elements of Petitioners’ cytokine-related arguments confuse association for causal effect, even though evidence cited for those arguments has scientific reliability. Fischer, for example, makes scientifically-reliable points about how LAIVs function. And articles like Vezzani *do* credibly suggest that certain proinflammatory cytokines, including IL-1 $\beta$ , are associated with seizure, and their findings are consistent with the arguments of Petitioners’ experts about neuron excitability and seizure threshold generally. But such literature also says little to nothing about peripheral stimulation of cytokines or their inevitable passage into the CNS—as opposed to cytokine generation *within* the brain or in *response* to existing seizure. These articles also do not establish that Flumist specifically would be expected to generate the particular proinflammatory cytokines identified by Petitioners’ experts—while other literature persuasively demonstrates it would *not*. Compare Fischer at 7 with Barria at 118, 120.<sup>39</sup>

Beyond the above, there are numerous other insufficiencies in the scientific reliability of the portion of Petitioners’ causation theory relating to cytokines. Petitioners’ experts did not persuasively establish that cytokines generated in response to Flumist would (a) likely travel into the CNS,<sup>40</sup> or (b) from outside the blood-brain barrier stimulate a response within, or (c) upregulate in sufficient amounts (and type) to impact a child with a hippocampal abnormality and thereby further lower his seizure threshold. At best, some of the literature filed post-hearing by Petitioners

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*Servs.*, No. 13–1002V, 2016 WL 3456436, at \*9–14 (Fed. Cl. Spec. Mstr. May 31, 2016) (infant's death not caused by cytokine upregulation due to vaccination), *mot. for review den'd*, 129 Fed. Cl. 176 (2016); *Cozart v. Sec'y of Health & Human Servs.*, No. 00–590V, 2015 WL 6746499, at \*6–7 (Fed. Cl. Spec. Mstr. Oct. 15, 2015), *mot. for review den'd*, 126 Fed. Cl. 488 (2016).”

<sup>39</sup> Admittedly, Barria (the only literature filed in this case directly considering the immunologic performance of Flumist specifically) involved samples taken from adults, arguably thereby reducing the weight it should be given since this case involves a three-year-old. Yet Fischer (relied upon by Petitioners) *also* only tested samples taken from adults. Fischer at 3. If one has no applicability to this case, then the same is true of the other (along with any other literature testing cytokines that did not involve pediatric subjects). Here, I find that the absence of studies directly involving the effects of vaccine-induced cytokines in the brains of pediatric subjects renders articles like Barria and Fischer the next best available proof, and thus worthy of some weight.

<sup>40</sup> Dr. Levin’s assertion that the mere fact that Flumist is administered literally “close” to the brain allows for the conclusion that it would somehow be “easier” for cytokines to travel into the brain was especially specious. Were he correct, than virtually *any* upper respiratory infection would similarly pose the threat of a drastic CNS-oriented injury.

establishes the different ways cytokines can travel into the brain. This does not establish that the receipt of a LAIV means it is more *likely* that this will occur, or that it will *inherently* occur in the absence of some coterminous infectious or disease process that encourages blood-brain barrier breach.

Petitioners' contention that cytokine activity in the brain would likely impact seizure threshold by heightening neuro-excitability, by contrast, was better supported by certain medical literature such as Vezzani. But this does not also mean that cytokines likely *instigate* that process—something even Vezzani seemed to recognize has not been causally demonstrated. Vezzani at 1287–88. And although Dr. Miller credibly established that the hippocampal abnormality that I.R.M. was discovered to possess has been associated with seizure, and also implicated in SUDC cases, Petitioners did not *also* establish with sufficient reliable evidence that seizures were more likely merely because a child received a vaccine that had the capacity to upregulate cytokines. Rather, they simply assumed this to be the case from the temporal relationship between vaccination and seizure, without any scientific or medical evidence that might demonstrate it.

Petitioners also could identify little literature specifically relevant to Flumist that connected its immune system-stimulating processes to seizure or SUDC. Thus, Fischer discusses LAIVs generally, and although it does support the conclusion that *some* cytokines are upregulated after receipt of a LAIV, it did not associate the vaccine with the specific cytokine (IL-1 $\beta$ ) most discussed by Petitioners' experts—and even seems to suggest (again, contrary to Petitioners' contentions that vaccination is only differentiable in degree from a wild infection) that the immune impact of the vaccine is wholly distinguishable from what a wild flu virus would accomplish. Meanwhile, other articles directly addressing Flumist, like Barria, did not support their claim.

In response, Petitioners have asserted in their post-trial brief that “the Vaccine Program has accepted that FluMist can cause systemic immune reactions,” citing to an older case purportedly standing for that proposition. Brief at 7–8, citing *L.A. v. Secretary of Health and Human Services*, No. 12-629V, 2016 WL 7664473, at \*13 (Fed. Cl. Spec. Mstr. Dec. 15, 2016).<sup>41</sup> But this is the *sole* such prior case involving seizure (there are no other similar cases involving SUDC after Flumist), so it is stretching things considerably to claim that *L.A.* reflects broad Program acceptance of Petitioners' contentions.<sup>42</sup> Moreover, *L.A.* is distinguishable in many regards. There,

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<sup>41</sup> Petitioners also reference the fact that cases involving Flumist have settled. Brief at 7–8. This of course is particularly non-probative support for their claim, since (a) cases settle for many reasons having nothing to do with the underlying claim's merits, and (b) settled cases do not result in reasoned decisions that provide any guidance in future circumstances. *Dillenbeck v. Sec'y of Health & Human Servs.*, No. 17-428V, 2019 WL 4072069, at \*13 (Fed. Cl. Spec. Mstr. July 29, 2019), *rev'd in part on other grounds* 147 Fed. Cl. 131 (Fed. Cl. 2020). While the fact a comparable case has settled before may give a petitioner “ammunition” in attempting to negotiate resolution of his own case, and may also offer hope that the claim has some meritorious elements, it does not guide a subsequent decision in any other meaningful respect.

<sup>42</sup> *L.A.* itself only references two other decisions regarding Flumist, neither of which involved a comparable injury. *L.A.*, 2016 WL 7664473, at \*13 (citations omitted). One involved Flumist working in concert with the human papillomavirus vaccine causing neuromyelitis optica, while the other caused acute hepatitis. *Id.*

a five-year-old who received Flumist after being taken to an acute care center to treat what appeared to be an existing viral infection subsequently suffered a fever followed by a seizure two days post-vaccination. *L.A.*, 2016 WL 7664473, at \*8. The special master in *L.A.* determined that the pre-existing infection likely interacted synergistically with the vaccine—a finding not possible herein, since there is *no evidence* I.R.M. had a fever or infection as of the date of his death. *Id.* at \*9, 16. And to the extent *L.A.* did determine that Flumist could cause a systemic immune response, that conclusion was not based upon the kind of evidentiary showing involving cytokine transport to the brain that Petitioners’ experts herein unsuccessfully endeavored to establish.<sup>43</sup>

Coloring the scientific and medical unreliability of theories offered in this case are deficiencies in the qualifications and testimony of two of the three experts who testified for Petitioners. Drs. Kinsbourne and Levin lacked testimonial credibility in important regards. Dr. Kinsbourne, for example, has no demonstrated research or treatment expertise in the matters in dispute, and he relies on neurology expertise that has not been honed or refined, whether by clinical practice or research, for nearly *30 years*. This is a criticism that has been lodged—reasonably—against him repeatedly in the Vaccine Program. *See, e.g., Pope v. Sec’y of Health & Human Servs.*, No. 14-078V, 2017 WL 2640503, at \*21 n.29 (Fed. Cl. Spec. Mstr. May 1, 2017). Although Dr. Kinsbourne may have a facility (drawn from his many prior turns as a Vaccine Program expert) in speaking to neurology topics with clarity and confidence, he is not *compelling or persuasive* in so doing—especially to the degree his statements are either not grounded in reliable science or do not arise from his own direct medical expertise or recent work.<sup>44</sup> The mere fact he has experience as a pediatric neurologist does not render him a helpful expert in *all* cases involving a pediatric neurologic injury.

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<sup>43</sup> I also note that *L.A.* does not clearly set forth the basis for its findings about the propensity of Flumist to produce systemic inflammatory responses that can promote a pathologic result, and thus may well have solely been the result of the special master’s acceptance of the expert opinions therein provided. This case, by contrast, involves a different set of experts and a very specific theory that overlaps somewhat with those rejected in prior SIDS cases. In addition, I find that Respondent’s experts made a persuasive showing that Flumist would *not* likely cause a pathologic upregulation of cytokines, or that those cytokines would necessarily cross the blood-brain barrier, and thereby interact with a brain malformation sufficient to lower seizure thresholds.

<sup>44</sup> Petitioners’ post-hearing brief notes (perhaps in the hope of vouching for his expertise) that Dr. Kinsbourne was the “only” neurologist who testified in this case—a true fact as far as it goes. Brief at 11. But this case turned mostly on questions of *pathology* and *immunology*—and on the latter topic, Dr. McCusker was a far more qualified immunologist than Dr. Levin, and provided testimony I found significantly more persuasive. *See also Copenhaver v. Sec’y of Health & Human Servs.*, No. 13-1002V, 2016 WL 3456436, at \*1 (Fed. Cl. Spec. Mstr. May, 31, 2016) (finding that Dr. McCusker was more credible than Dr. Miller), *mot. for review denied*, 129 Fed. Cl. 176 (Fed. Cl. 2016). In addition, the neurologic issues posed in this case related to the hippocampal abnormality, an issue that both sides largely agreed upon, disputing only the immunologic impact of the vaccine at issue, and Dr. Kinsbourne has no specific demonstrated expertise on that topic that would suggest his statements are deserving of extra weight. In any event, special masters are never bound to accept *any* expert’s statements as gospel—especially an expert who sees no patients, conducts no research bearing on the subject of his testimony, and cannot demonstrate otherwise any particular specialization in the relevant topic, as here.

Dr. Levin provides an even more glaring case of an expert out of his depth. Even if I ignore the fact that his daily life today revolves less around immunologic research than attorney work, it remains the case that his qualifications have been observed to be wanting in *numerous* Vaccine Program cases. *See, e.g., Bigbee v. Sec’y of Health & Human Servs.*, No. 06-663V, 2012 WL 1237759, at \*30 (Fed. Cl. Spec. Mstr. (Mar. 22, 2012) (stating that “Dr. Levin’s testimony in particular was extremely unhelpful—as would be expected from someone who practices law 99% of the time and thus medicine 1% and has not seen a patient since 1993”). And his testimony herein was conclusory and utterly unpersuasive. His credentials as an immunologist were far outweighed by Dr. McCusker’s, his pathology expertise overshadowed by Dr. Miller’s, and the assertions he made about the purported seizure-stimulating properties of innate immune system-produced cytokines were scientifically unreliable.

It is of course well-understood that in Vaccine Program cases, virtually *any* relevant evidence warrants some weighing and consideration, given that the Federal Rules of Evidence do not govern our proceedings. Section 12(d)(2)(B), (E); Vaccine Rule 8. And both of these experts had sufficient medical training to comment on the issues in dispute (although the same would be true for virtually *any* trained neurologist or immunologist). I therefore not only allowed into evidence Drs. Kinsbourne and Levin’s reports, despite my awareness of their experiential deficiencies, but read their reports and their supporting literature, and also listened carefully to their testimony at hearing. But none of the above means that these experts were automatically *credible* or *persuasive*. They were not, and their statements did not merit significant weight.

Dr. Miller, by contrast, was a more demonstrably-credentialed expert, whose base experience made him a good fit to comment on the issues in this case, and he provided cogent testimony setting forth Petitioner’s theories. However, although his expertise as a pathologist established a sound basis for opining on I.R.M.’s autopsy and brain tissue samples, his opinion about the theoretical impact of Flumist on a child with a possible susceptibility to seizure was unreliable (especially to the extent it arose from immunologic topics outside his admitted primary area of expertise). Moreover, and as Dr. Miller admitted, the opinion he provided overlapped somewhat with previously-rejected theories offered in SIDS cases, further reducing its persuasiveness in this case (even taking into account the differences between SIDS and SUDC). Tr. at 50–52. He ultimately could not ground his opinion in sufficient reliable scientific or medical evidence establishing the pathologic role of innate immune system-upregulated cytokines in the context of SUDC.

In short, the evidence offered in this case does not preponderantly support the conclusion that sufficient amounts of cytokines produced in immediate response to the Flumist vaccine could lower a seizure threshold in a child with the specified hippocampal abnormality sufficient to produce a death-inducing seizure, especially in the absence of a prior infection, fever, or known epilepsy. While it may be facially *plausible* that a vaccine administered close in time to an unexplained toddler death could have had some role in the outcome, the assertion is not scientifically *reliable* (given the evidence offered in this case).

## II. The Record Does not Support the Conclusion that I.R.M.'s Death was Vaccine-Caused

I.R.M. received the Flumist vaccine, and then showed no reaction beyond some lethargy the morning of September 26<sup>th</sup>. There is no evidence in the record that he ever had a post-vaccination fever. He was found unresponsive and in a prone position after his nap. His autopsy then revealed a hippocampal abnormality that both sides' pathologist experts deemed relevant to his death, and both proposed that a seizure was the likely immediate reason for that death. In addition, Drs. Miller and Harris agreed I.R.M. was probably dead for some time prior to his being found, although the precise time he died could not have been before the start of his nap.

Petitioners point to I.R.M.'s apparent pre-nap malaise as evidence of their theory at work. This is the sole evidence they can muster on this point, but it does not strongly support their theory. It is certainly a fair point to observe that vaccines can present transient symptoms reflecting an immune response is occurring (although Dr. McCusker's points about the *kinds* of cytokines that a LAIV would produce, along with the low likelihood that Flumist would generate certain proinflammatory cytokines stressed as causal herein, such as IL-1 $\beta$ , were not rebutted). And the statements of Mrs. Martin and her babysitter do suggest I.R.M. was not feeling well right before he napped. It is a reasonable inference to associate I.R.M.'s pre-nap condition with what thereafter transpired.

However, malaise is a somewhat nonspecific condition, especially for a young child, and it is equally likely based upon this record that I.R.M. merely felt tired.<sup>45</sup> More significantly, it is undisputed that I.R.M. was *not* running a fever prior to his nap on September 26<sup>th</sup>, thus greatly undermining the contention that IL-1 $\beta$ , which is associated with fever, was upregulated by Flumist—even assuming it *could be*, a conclusion that Dr. McCusker effectively rebutted. And I.R.M. displayed no other post-vaccination symptoms even within 24 hours of receiving Flumist (the period of time in which arguably cytokine production would at least have begun to peak, if Fischer or Kashiwagi are relied upon). So why was there not an earlier reaction? For Petitioners' experts, the malaise "proves" cytokines were interfering with I.R.M.'s brain, despite the absence of corroborative evidence that Flumist *in this case* caused sufficient upregulation of cytokines to produce outward clinical symptoms.

The record evidence about I.R.M.'s sleep in the days immediately after vaccination also undermines Petitioners' case. Certain experts like Dr. Kinsbourne placed great weight on the fact that I.R.M. had been placed down for a nap in the hours before he was found, suggesting that this

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<sup>45</sup> While I credit the joint statements of Mrs. Martin and the babysitter on this point, I also note that the babysitter contemporaneously informed first responders *both* that I.R.M. had seemed ok before his nap, and also that she checked him multiple times during the nap and found nothing wrong.

was somehow a unique factor that surely interacted negatively with his receipt of Flumist about two days before. But to make this argument is to disregard the fact that (as the record establishes) I.R.M. slept for two nights, without incident, before the nap at issue on September 26th. If sleep were so central to the circumstances of his death, then why did something not occur on either of the two prior nights? Petitioners' experts cannot answer this question simply by taking refuge in the rarity of vaccine injury generally.

At the same time, the record evidence *does* support an alternative explanation for the immediate trigger of I.R.M.'s death that Petitioners did not rebut. The police records establish that I.R.M. was found lying face down (and thus in a prone position) in vomit. Ex. 28 at 15; Ex. 55 at 15; Ex. 73 at 3. Dr. Harris credibly pointed out that I.R.M.'s sleep position, coupled with evidence of regurgitation, suggested that he had experienced respiratory failure, something that would be sufficient to trigger seizure in a child with the hippocampal abnormality I.R.M. possessed. Tr. at 193-95, 206. Sleep position is a *known* identified risk factor for SIDS. *Boatmon*, 941 F.3d at 1356; *Cozart*, 2015 WL 6746616, at \*8. It also has been correlated with SUDC cases. *See, e.g., Kinney II* at 213. Although SIDS and SUDC are not congruent concepts, they unquestionably overlap somewhat, and so sleep position is not irrelevant to the cause of death in this case. Petitioners' did not adequately address such other possible explanatory factors.<sup>46</sup>

The above paucity of evidence connecting Flumist to I.R.M.'s death underscores the overbreadth and circularity of Petitioners' causation theory. Petitioners have proposed a theory that, if accepted, would explain *any* post-Flumist death in a child with the same hippocampal deformity. Assuming the timing was medically acceptable, all that would be needed to provide the "did cause" evidence would be the fact of death itself, proving the purported cytokine invasion of the brain, and regardless of whether there is preponderant corroborative proof. But if so, then Petitioners have offered a self-realizing theory that relies on the "fact" of the alleged injury as proof of its cause. Such reasoning is not sufficient to meet the preponderant standard—and to require that Petitioners meet that standard is *not* "elevating" their burden, it is holding them *to the burden* set by the Federal Circuit. *Moberly*, 592 F.3d at 1322.

All in all, there is weak evidence here that I.R.M. was experiencing a proinflammatory reaction to Flumist due to cytokine upregulation as of the morning of his death. And although the pathologist experts on both sides in this case agreed that I.R.M. possessed an unknown hippocampal abnormality that likely played a role in these tragic circumstances, the record does

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<sup>46</sup> This is not a case in which the burden to prove alternative cause ever shifted to Respondent—and if it had done so, I would not on this record be able to conclude that the evidence for such an explanation preponderates for Respondent, since the record ultimately does not permit me to identify a likely cause of I.R.M.'s death. But I can and did consider this contrary confounding evidence when evaluating Petitioners' success in carrying their overall *Althen* burden. *Stone v. Sec'y of Health & Human Servs.*, 676 F.3d 1373, 1379 (Fed.Cir.2012) ("[o]ur decisions support the commonsense proposition that evidence of other possible sources of injury can be relevant not only to the "factors unrelated" defense, but also to whether a prima facie showing has been made that the vaccine was a substantial factor in causing the injury in question").

not preponderate in favor of the conclusion that the Flumist vaccine had anything to do with that seizure, by interacting with the abnormality or otherwise.

I also do not find that the record supports the conclusion that I.R.M.'s brain edema was vaccine-caused and/or the cause of his death. Although the record does establish that I.R.M.'s brain weight was large for a child of his age, it also supports the conclusion that the edema was attributable to his seizure and death rather than *causal* of it. Moreover, two of Petitioners' three experts seemed to abandon or ignore this theory at hearing (although it was included in counsel's post-hearing brief). The two most credible pathologists who testified, Drs. Harris and Miller, did not characterize edema as the cause of death, and it also finds no support in the autopsy or other medical records.

A case like the present illuminates what "logical sequence of cause and effect" really means, for purposes of preponderantly establishing the second *Althen* prong. There is no doubt that the theory offered by Petitioners' experts—that Flumist caused a proinflammatory cytokine environment sufficient to interact with I.R.M.'s previously-unknown brain abnormality and cause his death approximately two days later—can be stated so as to be temporally consistent with the basic facts herein, and thus literally provides the "logical sequence" required by *Althen* (418 F.3d at 1278). Based on similar reasoning, Program petitioners frequently maintain they have met their burden simply because their causation theory can be "fit" into the factual sequence pertaining to the claim.

But if all a petitioner need do is nestle his theory within the relevant fact pattern, what kind of preponderant showing has been made? What evidence from an injured party's actual circumstances suggest the vaccine likely "did cause" injury? Although the preponderant standard is not particularly onerous for non-Table claims, it still requires some "heavy lifting" by petitioners (*Hodges v. Secretary of Health & Human Servs.*, 9 F.3d 958, 961 (Fed.Cir.1993)) - not only to offer a reliable scientific theory, but also to show that the record *evidence* is consistent with that theory, and not just in a temporal sense. That kind of showing was not made in this case.<sup>47</sup>

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<sup>47</sup> In some cases, petitioners might support a successful *Althen* prong two showing with the statements or testimony of a treating physician having direct familiarity with an injured party's history. *See, e.g., Andreu*, 569 F.3d at 1375–77. Although it is often the case that such treater statements amount to little more than a narrative themselves, and do not always reference corroborative evidence substantiating the causal theory, they are still deemed worthy of weight because a treater who actually, contemporaneously dealt with the injured party *at the time of injury* is reasonably deemed to have insights into the facts of a case that an after-the-fact testifying expert can never possess. In this case, by contrast, there is no comparable proof. And in any event, no treater opinion is sacrosanct. Section 13(b)(1) (providing that "[a]ny such diagnosis, conclusion, judgment, test result, report, or summary shall not be binding on the special master or court"); *Snyder*, 88 Fed. Cl. at 745 n.67 ("there is nothing . . . that mandates that the testimony of a treating physician is sacrosanct—that it must be accepted in its entirety and cannot be rebutted").

### III. Petitioners' Motion to Strike is Granted

As noted above, Respondent chose to file a supplemental expert report from Dr. McCusker approximately four months after the hearing. He did so based on the assertion that Petitioners had themselves filed several “new” items of literature—although in fact, of the six articles filed after the hearing, *three* had been previously filed (and none were literally “new” otherwise, since all had been published *long before* the hearing—raising the question why the newly-filed articles were not offered in a timely manner). Indeed, Vezzani was not only previously filed but extensively discussed at hearing, and Kashiwagi was filed by *Respondent*. Petitioner moved to strike Dr. McCusker’s supplemental report.

Petitioner’s motion is granted. Not only had the formal deadline passed for the filing of expert reports in this case, but Respondent filed the supplemental report without leave—and without my requesting it either, as I did not indicate at hearing that I required any additional testimony from Dr. McCusker, who had ample opportunity to discuss cytokines and the degree to which they might cross into the brain as part of a pathologic process. While it is true, as Respondent argues, that Program claimants often file additional exhibits and evidence post-hearing, and while leniency in allowing such items into the record is generally appropriate, this does not mean that *every* filing out of turn must be accepted in all cases. If so, then the deadlines set by the special masters for acting in a matter amount to the mildest of suggestions that litigants are free to ignore.<sup>48</sup> In any event, this determination does not prejudice Respondent, since my Decision herein does not at all turn on the contents of Dr. McCusker’s supplemental report (and I have not considered it in deciding entitlement). I did not require additional comment from Dr. McCusker to comprehend the late-filed articles, or Petitioners’ ultimately-unpersuasive arguments about post-vaccination cytokine transport into the brain.

### CONCLUSION

The circumstances of this case are truly tragic. The Martins’s desire to identify the medical reason for the loss of their son is wholly understandable—as is their good faith basis for contending that the flu vaccine might have been involved, given the temporal proximity of its administration.

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<sup>48</sup> I give some credence to Respondent’s objections to Petitioners’ dilatory filing of three articles that were *not* inadvertent duplicates of previously-filed literature—although I have not stricken them, since they involve topics that both sides had full opportunities to address at hearing, and are otherwise relevant to the claim at issue. However, these three articles are not particularly helpful to Petitioners’ claim. They merely provide some ballast for Dr. Miller’s assertions about *ways* in which cytokines may cross into the brain – a contention largely not disputed by Respondent’s experts, although they may have quibbled about the precise application of different transport mechanisms to this case. They do *not* help Petitioners establish that the Flumist vaccine would stimulate cytokines with a pathologic potentiality relevant to seizures – or that the cytokines would likely have traveled into the brain in the absence of evidence of some other ongoing pathologic process, like an active infection or presence of some other toxic agent likely to allow easier permeation of the blood-brain barrier.

But the evidence offered in this case does not preponderate in favor of Petitioners' causation theory, and I cannot otherwise grant an award of damages simply because an injury occurred close-in-time to a vaccination. Thus, for the reasons stated above, I must deny entitlement in this case. In addition, Respondent's Supplemental Expert Report (ECF No. 79) is hereby **STRICKEN**.

In the absence of a timely-filed motion for review (see Appendix B to the Rules of the Court), the Clerk shall enter judgment in accord with this decision.<sup>49</sup>

**IT IS SO ORDERED.**

/s/ Brian H. Corcoran  
Brian H. Corcoran  
Chief Special Master

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<sup>49</sup> Pursuant to Vaccine Rule 11(a), the parties may expedite entry of judgment by filing a joint notice renouncing their right to seek review.